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BRITISH JOURNAL OF INDUSTRIAL MEDICINE

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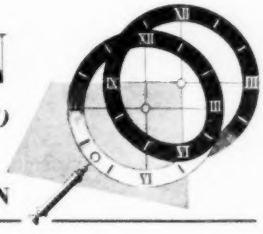

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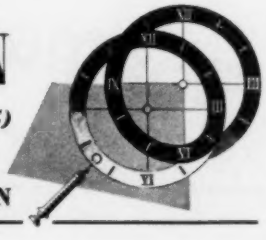

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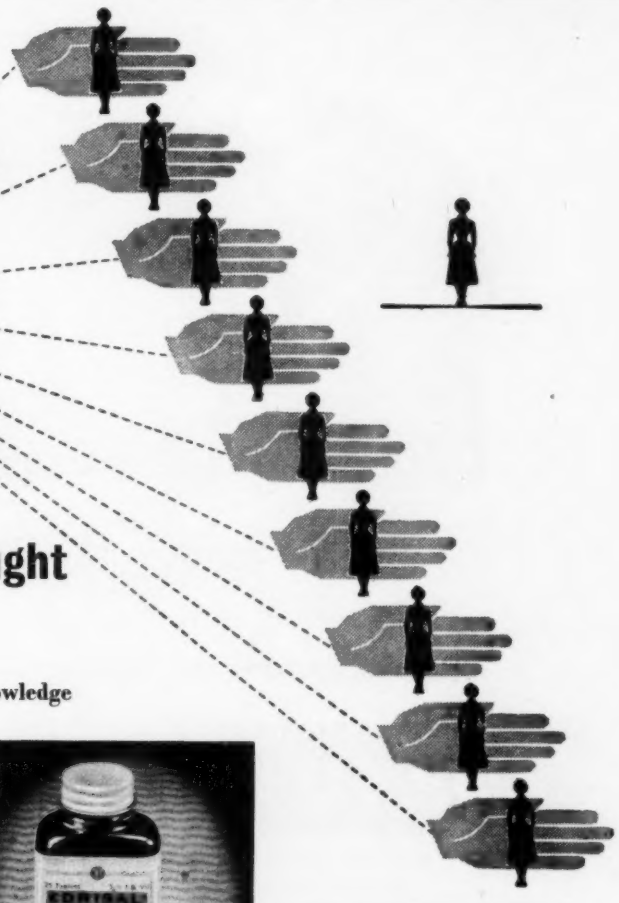

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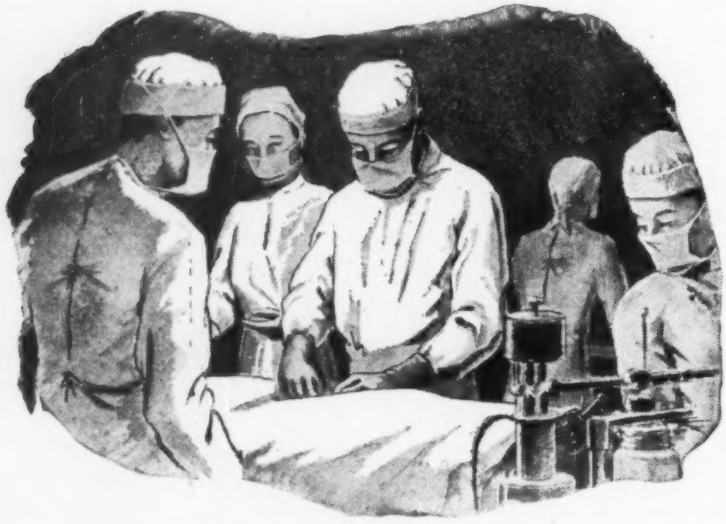

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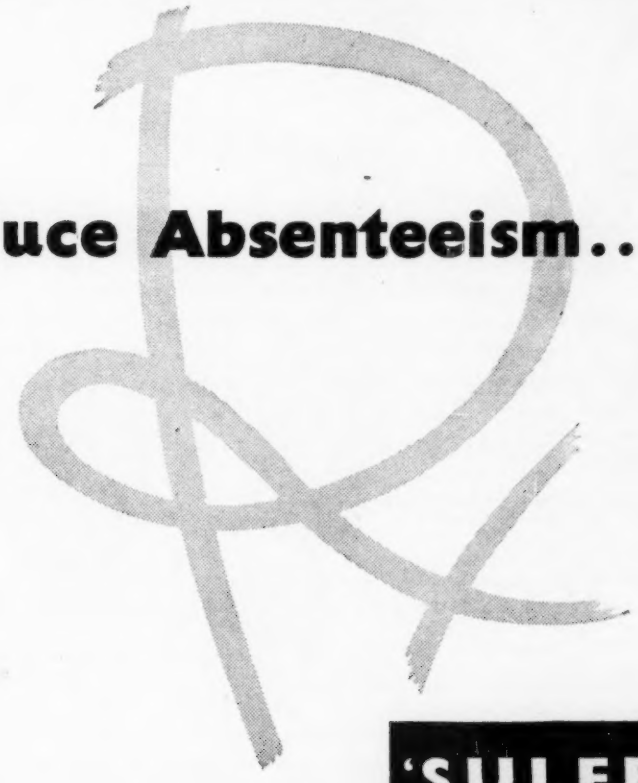

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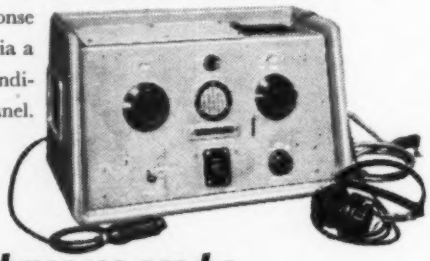

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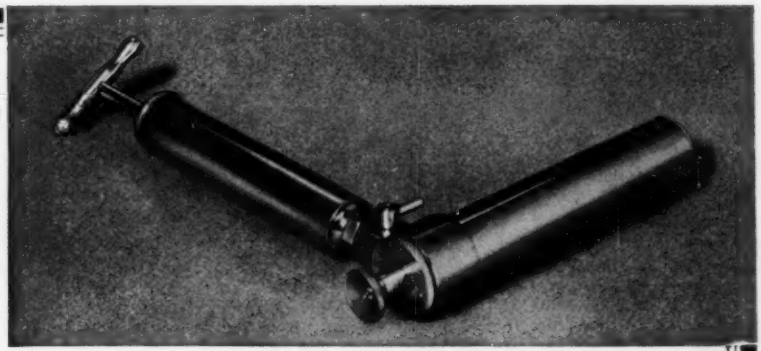

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BLOOD CHANGES IN INDUSTRIAL DISEASE*

BY

RONALD E. LANE

From the Nuffield Department of Occupational Health, Manchester University

I am honoured to be invited to give the second Ernestine Henry Lecture. I had the privilege of knowing this distinguished lady and of appreciating her interest in industrial medicine. She remained an inspiration to many and particularly to her son—Dr. Sydney Henry—whose work in this field is widely known.

A discussion of blood changes in industrial disease is justifiable for two reasons : all physicians should be aware of changes in the blood produced by toxic agents, and industrial physicians in particular should know how to make use of these changes in the medical supervision of workers. They must appreciate the limitations and sources of error of blood examinations. While full advantage should be taken of any objective method of measuring damage, it is important that figures used for this purpose are not endowed with false values. Blood changes may be considered separately as those that occur in the circulating blood and in blood formation. Poisons do not usually restrict their action to one organ nor are their effects likely to be restricted to one enzyme system in the blood. These changes are extremely complicated but with the growing knowledge of biochemistry more and more is being understood about them. For instance, though a poison may produce methaemoglobinaemia it is unlikely that this is the only change taking place in the cell. Less obvious and often more permanent changes occur ; a minor degree of haemolysis may be found and morphological changes, such as Heinz bodies and siderocytes, may result from the disturbance of the normal biochemical processes. Fig. 1 shows diagrammatically how certain industrial poisons affect the blood.

Changes in the Circulating Blood

Changes produced in the circulating blood range from simple pigment changes, as for instance when reduced haemoglobin is present in excess, to gross haemolysis produced by arsine. The pigment changes will be considered first.

Carboxyhaemoglobinaemia.—One of the best known of these pigment changes is that produced by the action of carbon monoxide. There is nothing to add to the extensive review by Killick (1940) except to draw attention to a recent recommendation that pure oxygen should be used in resuscitation rather than a mixture of oxygen and carbon dioxide which has been the practice for the last 30 years (Medical Research Council, 1952a).

Tolerance to repeated small exposures of carbon monoxide has been shown to occur in animals

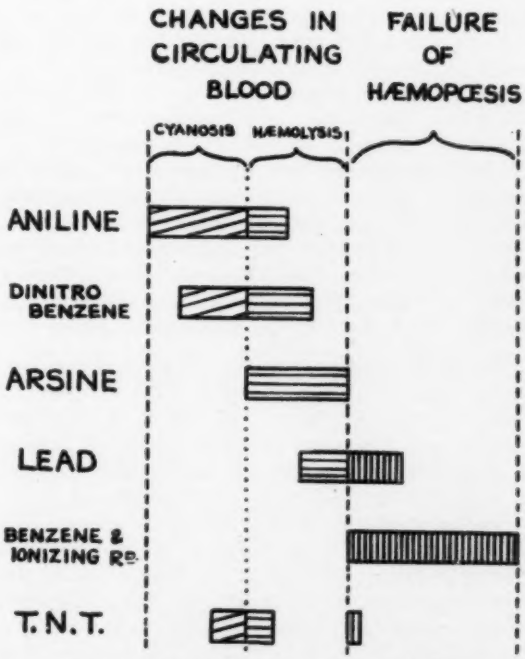

FIG. 1.—Effect of industrial poisons on the blood.

* Ernestine Henry Lecture delivered before the Royal College of Physicians of London on June 17, 1952.

which respond by an increase in the number of erythrocytes and in the haemoglobin level. Clinical evidence exists of a similar polycythaemia in men whose work exposes them regularly and frequently to carbon monoxide. Karasek (1911) records a red cell count of 9·68 millions in such a worker.

Methaemoglobinaemia.—Methaemoglobinaemia is the term used to describe the blood changes leading to cyanosis, caused by poisons producing an excess of unusual pigments. The pigment is often methaemoglobin but this is not always true. Sometimes sulphaemoglobin is responsible for the cyanosis, while occasionally methaemalbumin is also present.

Probably about 1% of the total haemoglobin in the red corpuscles is normally present as methaemoglobin. Reducing systems are present in the erythrocyte to keep all but this small percentage of the total haemoglobin active and they accomplish this unless the formation of methaemoglobin is abnormally high or they themselves become damaged. High concentrations of methaemoglobin are reduced at a rate of rather more than 10% in the hour. As the concentration drops, however, so does the speed of reduction. Methaemoglobinaemia occurs on rare occasions as a familial disease and is not uncommon as the result of the toxic action of certain drugs such as the sulphonamides.

Methaemoglobinaemia in industry is usually the result of the absorption of the nitro and amino derivative or compounds of the aromatic series. There is a great variety of substances in this group and their number is continually increasing. Many of these compounds are in that large group of industrial poisons which are readily absorbed through the skin and respiratory tract. Toxic substances which can gain access to the body by both these routes are likely to present serious health problems. Table 1 gives some of the compounds which produce methaemoglobinaemia.

Aniline Poisoning.—Aniline is a good example of this group and has given its name to the condition of cyanosis widely known as "anilism". Methaemoglobinaemia following the absorption of aniline is caused by the products of its metabolism, such as phenylhydroxylamine and p-aminophenol.

Methaemoglobin cannot transport oxygen, so in anilism there is a decrease in the available arterial oxygen. Moreover the dissociation of the remaining active haemoglobin is further impaired in accordance with the well known Haldane effect. Despite this, workers with obvious cyanosis due to the formation of methaemoglobin may be little

TABLE 1

SUBSTANCES PRODUCING METHAEMOGLOBINAEMIA

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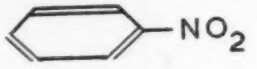
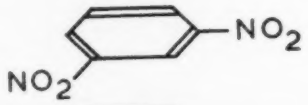
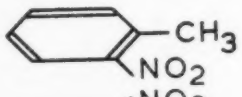
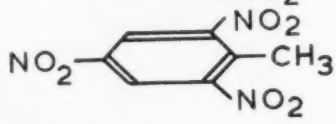
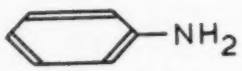
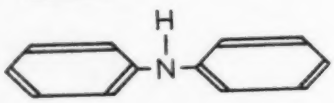
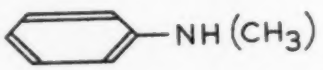
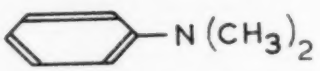
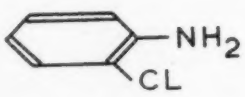
Nitrobenzene	
Dinitrobenzene	
Nitroaniline (esp. P)	
Mononitrophenol (o.m.p.)	
Nitrotoluene o.m.p.	
Trinitrotoluene (2:4:6)	
Amino Compounds	
Aniline	
Diphenylamine	
Methylaniline	
Dimethylaniline	
Chloraniline	

the worse, and may not suffer from obvious dyspnoea. It is only when the methaemoglobin reaches levels of about 40% that dyspnoea on exertion occurs.

In more severe exposure, usually following accidental skin contamination, the patient becomes pale and then cyanosed. He complains of a fullness in the head and of a bursting, throbbing

headache. He feels weak and weary, sometimes he vomits and complains of abdominal cramps. Very heavy exposure is followed by a fall in blood pressure, rapid breathing, coma, and death. Such catastrophes are happily rare. Successful treatment of acute cases, as with all poisons which are absorbed through the skin, depends on the immediate removal of soiled clothing and on cleaning the skin. In the old days the workman, having been re-clothed, was put into an open car and driven furiously round the countryside. The purpose of this robust treatment is not clear, but it has now been superseded by giving methylene blue which is very effective in true methaemoglobinaemia. It should be given in doses of 1–2 mg. per kg. of body weight ; 10 ml. of a 1% solution in normal saline are usually sufficient for the ordinary man. Both theoretically and experimentally the value of oxygen in the treatment of pure methaemoglobinaemia is doubtful, so that time and effort should not be wasted in giving it.

In the more chronic forms of aniline poisoning the blood changes are those of haemolysis and anaemia. Erythrocytes show stippling and there is evidence of marrow hyperactivity.

Dinitrobenzene after severe exposure produces methaemoglobinaemia ; when exposure is small and repeated it produces a marked anaemia, presumably because the red cells have been damaged. This is probably additional to the pigment change which has always been regarded as being reversible, though even in aniline poisoning Crick and Jackson (1952) believe that although the methaemoglobinaemia clears rapidly the erythrocytes are changed for the remainder of their life.

It may well be that these chronic changes are due to the formation of other breakdown products. Barkan and Walker (1940) and Lemberg, Legge, and Lockwood (1941) have independently advanced the hypothesis correlating the formation of choleglobin with the breakdown of haemoglobin, the formation of Heinz bodies, the destruction of reducing enzymes, and the haemolysis of red cells. From what has been said it becomes clear that poisons which produce these pigments are likely also to produce anaemia and morphological changes in red cells.

Trinitrotoluene Poisoning.—The cyanosis after absorption of trinitrotoluene is usually due to methaemoglobinaemia which develops slowly and may be present for prolonged periods with few obvious ill effects. In the early days of the last war, when work was being done in munition factories under bad conditions, large groups of workers sometimes showed the dusky complexion of T.N.T. cyanosis. They seemed little the worse for the methaemoglobinaemia but it is certain that this level of absorption was such that the more susceptible would eventually show other manifestations of T.N.T. poisoning. In a small percentage of these patients the pigment responsible for the change of colour was sulphaemoglobin (Jope, 1946). A slow reconversion of methaemoglobin to normal takes place, but this does not happen with sulphaemoglobin and the pigment remains as long as the erythrocyte remains intact.

Like the other substances producing pigment changes T.N.T. is a mild haemolytic poison and this is by no means confined to those patients who are cyanosed. Stewart, Witts, Higgins, and O'Brien (1945) observed a fall in haemoglobin concentration and in the red cell count in students exposed to T.N.T. Their work suggested that attempts by the bone marrow to compensate for this are inhibited and only begin to become fully effective 48 hours after removal from contact. These observations were carried out during the first five weeks of exposure to a concentration of T.N.T. varying from 0·3 to 1·3 mg. per 10 c.m. of air. My own clinical experience supports the suggestion of Stewart and her colleagues that after a time tolerance to T.N.T. is acquired and haemopoiesis returns to its normal level.

Blood examinations of T.N.T. workers are still carried out. Haemoglobin and red and white cell counts are thought to give useful information, but a critical appraisal of their value is needed.

Haemolytic Poisons.—One of the best examples of a haemolytic poison is arsine which can cause rapid death. It is given off when nascent hydrogen comes into contact with arsenic. The hydrogen is produced by the action of acid on a metal and the arsenic is usually present as an impurity. Locket, Grieve, and Phillips (1952) have recently reviewed the literature which includes reports of poisoning in a wide variety of industries such as electroplating and the smelting, refining, and galvanizing of metals. Dudley (1919) has reported arsine poisoning in the crew of a submarine. In the presence of oxygen arsine reacts with haemoglobin to form choleglobin and other pigments and the arsenic becomes fixed in the erythrocyte until haemolysis occurs. Where poisoning is severe this begins

The method of preserving specimens of blood is important. Oxalated blood may be used for the estimation of carbon monoxide but, where methaemoglobin or similar pigments are to be estimated, sodium fluoride should be used to preserve the specimen by inhibiting glycolysis. The blood should be collected in a blood sugar bottle and kept in the dark. Air should be excluded by filling to the stopper with liquid paraffin and the estimation should be made as soon as possible and not delayed more than five hours.

to take place within about six hours of inhaling the gas. Early symptoms are headache, nausea, epigastric pain, vomiting and diarrhoea, and in about 10 hours some red or black urine may be passed. In 24 hours the skin may have the characteristic coppery colour. The red cell count may have dropped to one million and the haemoglobin to 20% (3 g.). If the patient recovers the anaemia sometimes persists due to depression of haemopoiesis but on the other hand the haemoglobin level may rapidly return to normal. Immediate oliguria is usual and in severe poisoning there is complete suppression of urine. It seems likely that death is due to renal damage and where this is less severe the patients survive. Treatment should be directed to maintaining the body fluids as near normal as possible. Oxygen should be given freely and slow transfusion of packed cells is of value if the anaemia is severe. B.A.L. is of doubtful value but may be of use if the condition is diagnosed within a few hours.

Lead Poisoning.—Lead is an extensively used toxic material. In lead poisoning there is a low grade hypochromic and normocytic anaemia rarely as severe as 60% (9 g.) with a fall in the number of red cells, with marked punctate basophilia, and with less constant changes in the leucocytes. The earlier workers attributed the anaemia of lead poisoning to haemolysis. Aub, Fairhall, Minot, and Reznikoff (1926) explained this by postulating a change in the red cell which made it more brittle and less able to withstand the trauma of circulation. The rise in reticulocytes was regarded as the normal response to this rapid destruction of circulating red cells. But Rimington (1938) advanced the hypothesis that the anaemia was due to a different cause, namely the blocking by lead of the entrance of iron into the protoporphyrin ring ; he explained in this way the excretion of coproporphyrin III in such large quantities in lead poisoning. He discounted the haemolytic effect of lead since faecal urobilinogen was normal. Many aspects of haemopoiesis in lead poisoning still remain obscure. In 1942 Kench, Gillam, and Lane suggested that the excretion of coproporphyrin III was not sufficient by itself to account for the degree of haemoglobin deficit and suggested that lead interfered all along the line of enzymatic synthesis causing restricted formation of porphyrin and other intermediates.

McFadzean and Davis (1949) reconciled these newer theories of deficient haemoglobin formation with the older one of haemolysis. They studied the effect of splenectomy in rabbits poisoned with lead, and concluded that the faulty haemoglobinization was associated with stippled red cells which were removed from the circulation by the spleen, producing a haemolytic type of anaemia. We have recently studied seven cases of lead poisoning (Kench, Lane, and Varley, 1952) and followed their coproporphyrin excretion and have shown that not only is coproporphyrin III greatly raised but also coproporphyrin I. This pigment is excreted in excess in haemolytic conditions characterized by hyperplasia of the bone marrow. These results support the view that lead is both a haemolytic and haemopoietic poison.

Changes in the white cells in lead poisoning have been described by numerous workers including Legge and Goadby (1912), Brookfield (1928), Ferguson and Ferguson (1934), and Shiels (1950). The most constant finding is an increase in the ratio of monocytes and large lymphocytes to small lymphocytes. The Fergusons found this change in ship breakers who were absorbing lead, while Shiels, working in Australia, found similar changes in a series of 400 workers employed in various lead trades.

In the medical supervision of lead workers blood examinations have long been used and there is certainly a place for a simple test which can be made by the examining doctor. In France routine periodic red cell counts are used to assess lead damage, and are required by law. It is doubtful, however, whether red cell counts are useful even in the more heavy exposures and certainly in modern industry, where exposures are far less than in the past, they have little value. I have found white cell changes of little value in the routine control of lead workers. McCord, Holden, and Johnston (1935) advocated the use of a basophilic aggregation test which enumerates both stippled cells and reticulocytes. I prefer the use of punctate basophil counts, provided they are done under standard conditions, in the medical control of lead workers, and I have previously discussed their value (Lane, 1949).

More recently the value of coproporphyrin excretion in the urine has been considered. There is little doubt that the level of excretion gives an early indication of the effect of lead on the blood and blood-forming system. Changes in excretion occur very early and before the appearance of other manifestations of lead poisoning. The ranges of coproporphyrin excretion of a small group of workers who had been heavily exposed to lead are shown in Fig. 2. It will be seen that urinary samples may be misleading if taken alone. As a simple method of medical control, I still prefer the punctate basophil count and haemoglobin

estimation, but urinary coproporphyrin levels are of value provided they are used for groups and not single persons. Nevertheless, none of these examinations can wholly replace the estimation of lead in the urine.

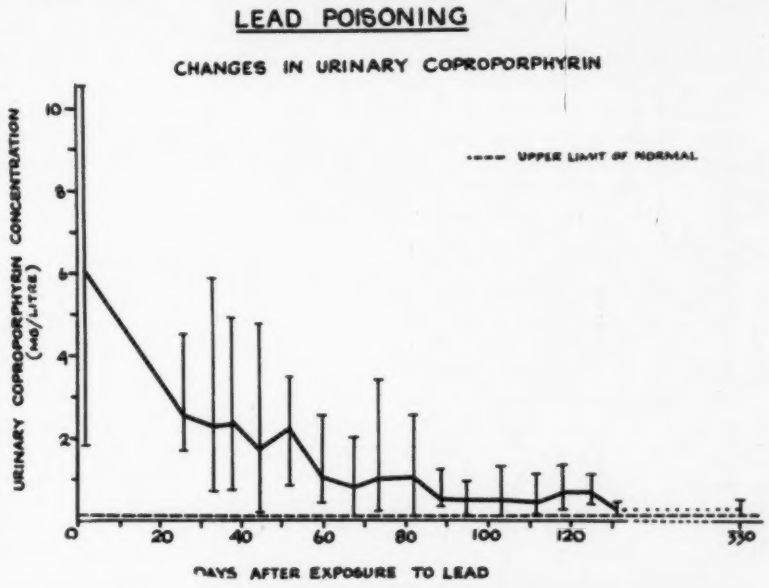


FIG. 2.—Changes in urinary coproporphyrin excretion of six men with lead poisoning. Vertical lines give the ranges of observations.

Changes in Blood Formation

Industrial poisons producing changes in the peripheral blood have so far been discussed, although in lead poisoning there is also an interference with blood formation.

There may also be aplastic changes in T.N.T. poisoning. Fatal cases of aplastic anaemia among T.N.T. workers were reported in this country in both world wars. Five T.N.T. workers who suffered from aplastic anaemia came under my care in the last war. In all there was profound anaemia, leucopenia, and thrombocytopenia. In one patient the red blood cells were below one million, the absolute polymorph count was under five hundred, and he had many haemorrhages. With repeated transfusions he recovered and is now the father of a large family. This is the first case so far as I can find of a severe T.N.T. aplastic anaemia to recover (Fig. 3). With similar treatment all five of these patients recovered and have been followed up to the present time, and none has shown any indication of being any the worse for these grave episodes. The blood changes during treatment are illustrated in Figs. 4 and 5 for two other of these patients.

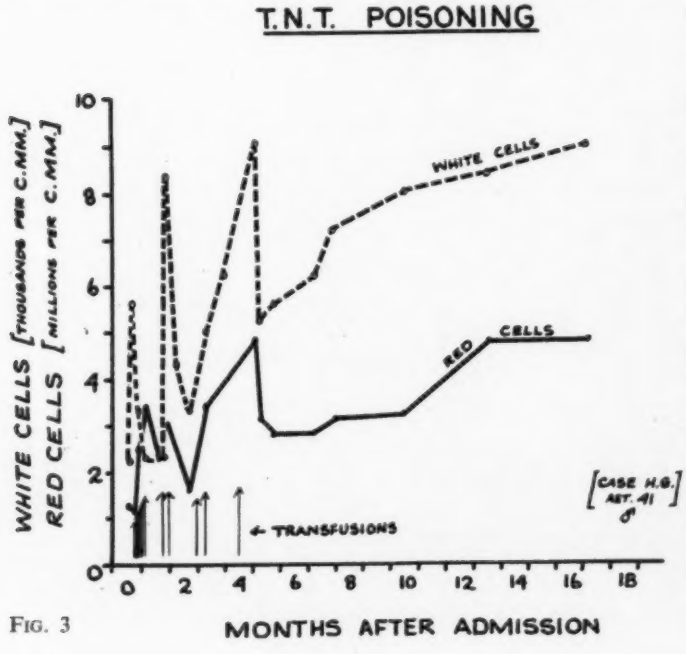


FIG. 3

The clinical picture before treatment is identical with benzene poisoning but the prognosis is quite different. I have yet to see a patient, suffering from benzene poisoning with severe aplasia and haemorrhages, recover, though transfusions may carry them on for a year or two.

The most important haemopoietic changes, however, arise from exposure to benzol and ionizing radiations.

Benzene (Benzol).—Benzol poisoning provides an interesting chapter in industrial toxicology and its vast literature has been reviewed by Browning (1937). The term benzol is a more satisfactory one than

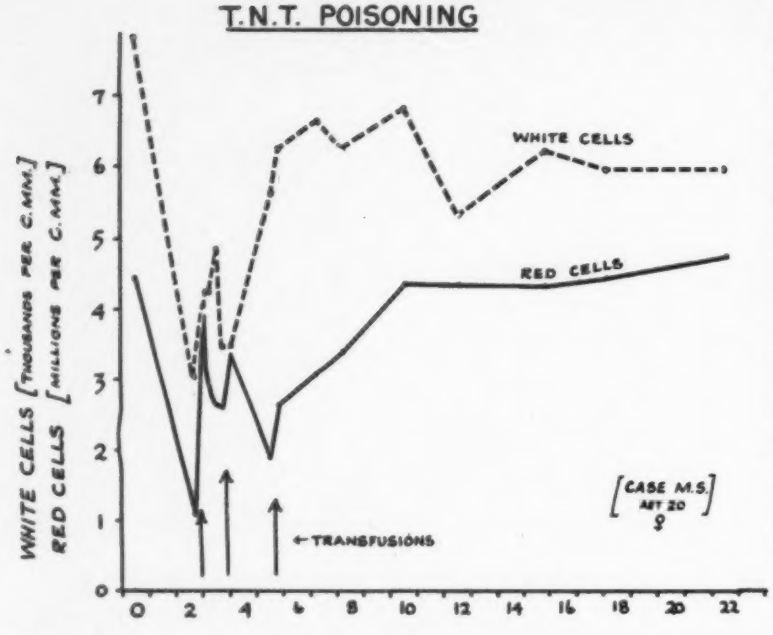

T.N.T. POISONING
WHITE CELLS [THOUSANDS PER C.MM.]
RED CELLS [MILLIONS PER C.MM.]
WHITE CELLS
RED CELLS
[CASE M.S. AET 20 ♀]
←TRANSFUSIONS
0 2 4 6 8 10 12 14 16 18 20 22
MONTHS AFTER ADMISSION

FIG. 4

benzene because in industry pure benzene is rarely used. The commercial product (benzol) contains toluene and xylene as well as various other impurities in smaller amounts. This mixture of simple volatile substances can produce profound and long-term effects which are almost entirely confined to the blood. Schrenk, Yant, Pearce, Patty, and Sayers (1941) showed that the distribution of benzene in the body follows physico-chemical laws which account for the high concentrations in fat and bone marrow; these tissues contain some twenty times as much benzene as the blood. It may well be that this partition of benzene between water and fat is one factor which determines the selective action of this poison on blood formation, though its mode of action is still obscure.

Benzol is widely used in industry. The main dangers arise in its use as a solvent, in producing rubber cement, in the leather cloth industry, and in quick-drying inks. In this type of work it is often used under conditions which cannot be easily controlled. In the chemical industry, particularly in the dyestuffs section, it is used widely but here, as in its distillation from coal or coal tar, it is more readily controlled by complete enclosure. For over 50 years benzol has been known to be a dangerous poison yet this is still not appreciated by many employers and industrial chemists. Continuous daily exposures are more dangerous than single heavy exposures and there is no agreement as to what constitutes a safe limit. The Safety Code of the International Labour Office (1949) lays down 35 p.p.m. as a safe limit but some workers suggest that it is unsafe to allow any measurable quantity in the atmosphere which is to be breathed continuously for eight hours a day. The usual clinical

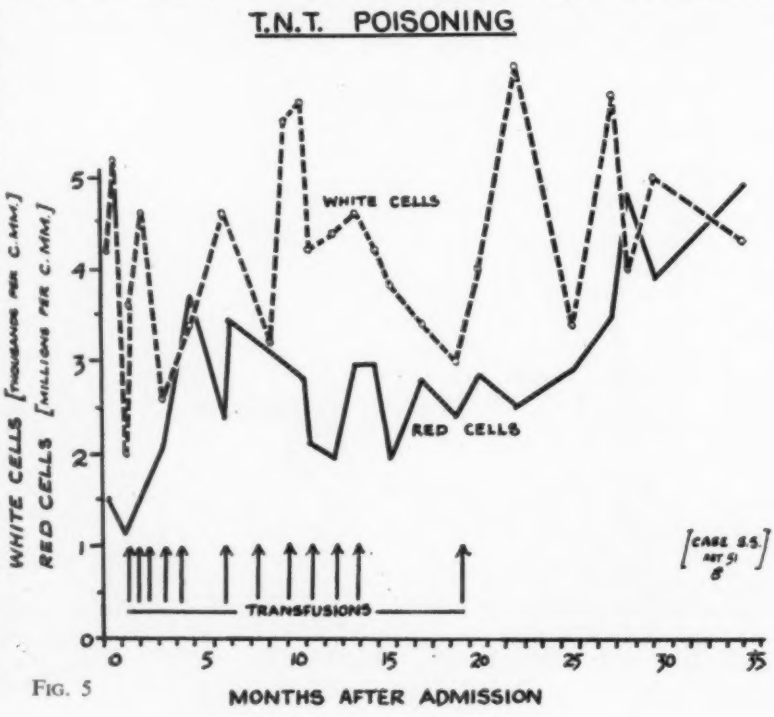


FIG. 5

picture of benzol poisoning, aplasia with anaemia, leucopenia, and widespread haemorrhages, is frequently described. Further study, however, over the last 20 years has shown a great diversity in the manifestations of this poison. The examination in the U.S.A. of groups of workmen exposed to benzol by Hunter (1939), Greenburg, Mayers, Goldwater, and Smith (1939) as well as the pathological studies of Mallory, Gall, and Brickley (1939) have all added considerably to our knowledge. The effect of benzol on the marrow may be to produce a picture of heightened or reduced activity and these changes will be reflected in the circulating blood.

Not only are many of the descriptions of benzol poisoning that appear in textbooks out of date but the official description for the purpose of notification of industrial poisoning (Factory Department Form 1787, 1925) is no better, for it states that benzene produces " a severe form of anaemia with haemorrhages under the skin and bleeding from the gums, nose, and other passages in the last stages ".

Some of the atypical cases are notified under the term " toxic anaemia ", but a description of the variety of blood changes which it can cause would help in its early diagnosis and in the detection of the atypical cases which have been described by Bousser, Albahary, and Tara (1952) in France. Chronic benzol poisoning has for some time been recognized as a cause of leukaemia (Erf and Rhoads, 1939), more usually myeloid but sometimes lymphatic.

The variety of blood changes now accepted as being caused by benzol may be due to the fact that atypical poisoning was not recognized in the early years of the century, or that the mixture of substances now sold as commercial benzol has changed and this in turn has changed the clinical picture of the disease. The doctor responsible for the care of benzol workers should also be familiar with the early blood changes. In this connexion the work of Goldwater (1941) is valuable because he observed a number of workers over a long period and was able to study a comparable group not exposed to benzol. He found that among a group exposed to 11–1060 p.p.m. for a period of from six months to six years the abnormalities most frequently observed were anaemia, macrocytosis, and thrombocytopenia. Leucopenia was present in only a small percentage of the men but lymphopenia, both relative and absolute, was a common finding. The haemoglobin values were relatively high. It is significant, however, that of those patients showing severe blood changes, all had leucopenia, three-quarters had thrombocytopenia, and half a reduced red cell count.

One of the striking features of benzol poisoning is the occasional delay in the onset of symptoms which may be as long as two years after exposure has ceased. Minor changes would probably have been detected had a careful search been made, but these patients appear to remain well for one or two years until perhaps some severe infection or other form of stress supervenes. There are considerable variations in susceptibility to benzol and cases are recorded where minute daily doses have been fatal, and there are others which have been exposed for years to potentially dangerous concentrations with no obvious ill effects. There is no conclusive evidence that women are more susceptible than men. On general principles, however, young people and pregnant women should be prevented from coming into contact with benzol, and my own experience suggests that those over 60 may be less resistant than younger adults.

A poison so widely used and capable of producing such a variety of blood changes presents a difficult problem in medical supervision. Routine blood examinations must be as simple and as rapid as possible. A leucocyte count used to be considered sufficient, and when it fell below 5,000 per c.mm. the worker was removed from exposure. The wide variation of the normal white cell count makes the interpretation of minor changes difficult. Complete red and white cell counts with differential counts should be done when possible. Browning (1952), who has considerable experience of this disease, recommends total white cell counts with differential counts. She is emphatic that a decrease in the polymorphs is the most significant early sign of benzol poisoning.

Two men suffering from benzol poisoning, referred to me by H.M. Medical Inspector of Factories, illustrate some of the points mentioned. They worked in a small leather cloth factory in which some 40 men had been exposed to benzol for 10 years before 1949. The extent of exposure varied but at least 12 men, including the two cases described, were approximately equally exposed.

F. M., a man aged 65, had been employed for seven years as an assistant spreader in the manufacture of leather cloth. He was first observed by one of H.M. Inspectors of Factories in 1948 when he had a considerably reduced polymorph count of 2,000 ; at two further examinations in 1949 the counts were 2,000 and 550. In May, 1950, when he was first seen in hospital he had a red cell count of 4½ million and a white cell count of 3,500 with 11% polymorphs. The marrow at this time was generally depressed, the myeloid series being mainly affected. Three months later his red cell count fell

rapidly to 1·5 million with 30% haemoglobin (4·4 g.). His white cell count was 1,000 with 8% polymorphs (Fig. 6). Repeated transfusions failed to help him and he died in December, 1950.

J. L., a man aged 67, worked side by side with F. M. and was employed for a similar time. He was first seen by me in January, 1951. He had worked almost continuously as a spreader since 1941, but in 1949 the use of benzol was stopped. He had a large liver and spleen and a white cell count of 50,000 with 90% lymphocytes—the typical picture of chronic lymphatic leukaemia. Later counts have reached 200,000 lymphocytes. He has had various forms of treatment and the disease appears to be taking its usual course.

The use of A.C.T.H. in the treatment of toxic aplasias must be considered. The M.R.C. panel (1952b) has given little encouragement to such therapy in the idiopathic aplasias, but in their report the patients who showed most improvement from this treatment were the toxic cases of thrombocytopenia. Hart, Wraith, and Mansell (1952) record the successful treatment with A.C.T.H. of a patient with agranulocytosis due to drugs. This and the records given of two similar cases suggest that A.C.T.H. should be tried in aplastic anaemia due to benzol.

Toluene and xylene are widely used as a substitute for benzol. These two substances with a much higher boiling point and lower vapour pressure are much safer than benzol. Not only are they less volatile, but they are metabolized in a different way. In an investigation by Von Oettingen, Neal, Donahue, Svirbely, Baernstein, Monaco, Valaer, and Mitchell (1942), of the U.S. Public Health Service, it was found that toluene had no effect on blood-forming tissues in animals.

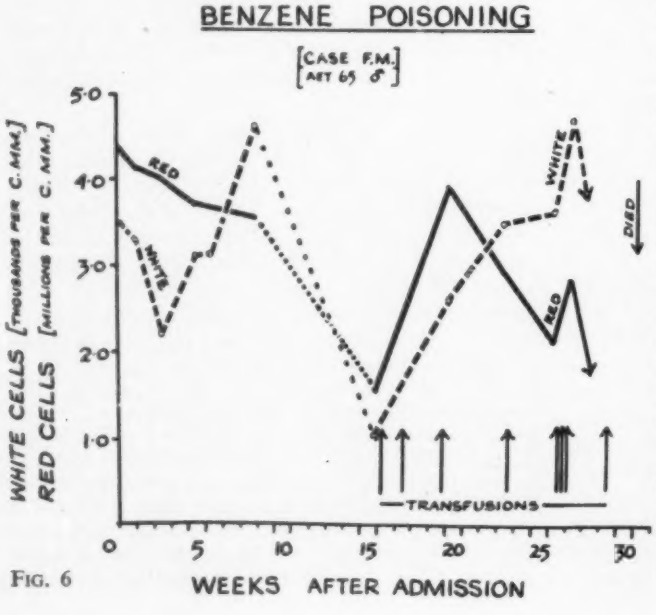


FIG. 6

Ionizing Radiations.—Small repeated doses of ionizing radiations may cause a depression of the bone marrow and the reticulo-endothelial system, giving rise to a leucopenia, sometimes preceded by a transient leucocytosis. This is often accompanied by a lymphopenia and if allowed to progress an aplastic anaemia may develop. As with benzol, leukaemia sometimes follows. A high incidence of leukaemia has been reported among radiologists by Levent (1932) and more recently in Japan among the populations who were exposed to the two atomic bomb explosions (Warren, 1952). The safe limit of exposure, which is now taken in Great Britain as 0·5 r (roentgen) per week, is constantly under review. It is important to know as accurately as possible the lowest exposure which can produce long-term effects.

The number of processes in which workers are exposed to ionizing radiations is rapidly increasing. Powerful *x*-ray machines are used to detect flaws in castings and welds, and radium is used for similar purposes. Luminizing—which involves painting dials with a paint containing minute quantities of radium—has been done for many years. The manufacture and use of fission products in atomic energy factories has introduced a new industrial hazard, and many laboratories employ tracer techniques which may be hazardous to the chemists concerned. Much is done both in industry and in hospital to reduce exposure to dangerous radiations to a minimum, and great strides in protection have been made, particularly in the design of *x*-ray apparatus. There are now probably few radiologists and industrial workers whose hazard is as great as that of the nurse who is looking after a patient being treated by radium.

In industry the possible danger of luminizing was recognized soon after the 1914–18 war. Several years after exposure had ceased a number of young women died of bone sarcomatosis (Martland, 1931). At the beginning of the last war, when a great increase in this work appeared likely, regulations were introduced in this country and arrangements were made for a clinical and haematological examination by a medical inspector of factories. The results of this work are recorded by

Browning (1949). She concludes that, working under prescribed conditions, luminizers over a period of five years have shown no evidence of a depression of the bone marrow. She did, however, notice a slight hyper-stimulation. It was possible to follow up these girls for periods of one to four years after exposure ceased and these examinations showed that a complete reversion to normal had taken place. Browning concludes that the hyper-stimulation of the reticulo-endothelial system, suggested by the " high normal " white cell count, the relative lymphocytosis, and the appearance of young forms of the monocyte, is caused by inhalation of small amounts of radioactive material, and that it is transient only, since these changes disappeared after exposure had ceased. The immense labour involved in keeping this group of workers (at one time as many as 540) under review—scattered as they were throughout the country—is amply repaid by the evidence it has produced, namely, that luminizing is harmless if carried out in accordance with regulations.

The medical control of workers using *x*-rays has depended mainly on blood examinations. The British *X*-ray and Radium Protection Committee for *X*-ray and Radium Workers (1948) recommend an initial complete blood examination with rejection if the white blood count falls below 5,000 and if the lymphocytes are below 1,500, and repeated total and differential counts every three months. This standard has been widely adopted and in large Government factories, where processes involve exposure to ionizing radiations, blood examinations are extensively used. As a result considerable advances in knowledge are taking place, and Edson and Williams (1952) have recently suggested a modified standard. They regard a white cell count of 4,500 with 3,000 polymorphs or 1,000 lymphocytes as a warning level, at which men may continue to work but should be under strict supervision. A white cell count of 3,000, with 2,000 polymorphs or 700 lymphocytes, or a film in which numerous immature cells are constantly present, calls for rejection from work involving exposure to ionizing radiation.

An investigation was carried out at the Atomic Energy Research Establishment by Chamberlain and Turner (1951) to determine the reliability of white cell counts. Statistical evaluation of the counting techniques, which were standardized, showed a coefficient of variation of under 10% in leucocyte counts and 12–15% in neutrophil and lymphocyte counts. With extra special care the coefficient of variation for the total white count could be reduced to 5%.

Both daily and seasonal variations in the white cell counts were noted. Averages for counts in the winter were some 15% higher than for counts in the summer. Exercise and emotion as well as infection produce transitory changes in the leucocyte count. In much of the routine work that has been done in the past some of these normal changes have been ignored and false values have been attached to many of the blood counts made. To have any meaning, white cell counts should be done on men at a standard time of the day and they should not follow soon after severe exertion, a meal, or emotional disturbance. The initial count, before the man starts work, must often perforce ignore these restrictions. (He has often, for instance, cycled a considerable distance and is almost invariably under nervous stress.) It is not surprising, therefore, that the controls as well as workers exposed to radiation show a significant fall in the white count following this first examination. The authors conclude that in view of all these factors the six-monthly white cell count does not in fact afford the best protection to workers exposed to very low doses of radiation. They maintain that better protection is likely to result from a film badge which measures the exposure of the whole body to radiation. They advise the suspension of the routine six-monthly blood examinations of the many thousands of workers who are theoretically exposed to radiation, but recommend that a more frequent check is maintained on the smaller number who are exposed to larger doses.

Most experienced workers in this field would agree that where the total internal and external radiation does not exceed 10% of the maximum permissible level, routine blood examinations may be dispensed with. There is, however, much yet to be learned about this subject, and if such a policy is adopted the precaution should be taken of examining over a long period (10 or 20 years) representative samples of those exposed to low levels of radiation, and in this way detect any long-term effect. For those whose exposure is greater haematological control is generally regarded as advisable. It is as yet too early to pronounce with any finality on the nature and frequency of these blood examinations. It seems probable that a white cell count with a differential count is likely to provide the most important information with the least effort, but more knowledge is required of the significance of these blood changes.

Conclusion

The value of blood examinations in the medical supervision of workers exposed to certain hazards

has been discussed. It is important that facilities for such examinations should be more widely available. Many of the large factories have set up their own haematological laboratories, but when the hazards arise in small factories or in small groups of workers no such outlay can reasonably be expected. These examinations should be undertaken by well-equipped and well-staffed laboratories of the regional hospital boards, or by industrial health laboratories established to do this work. In this way, and in this way only, can the present knowledge of blood changes in industrial disease be used to prevent disease instead of leaving our medical services helpless to deal with the end-results.

I have pleasure in acknowledging the debt I owe to members of my department in Manchester and to many other friends and colleagues for the help and criticism they have given me in preparing this lecture.

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THE CARCINOGENIC ACTION OF 4-AMINODIPHENYL AND 3:2′-DIMETHYL-4-AMINODIPHENYL

BY

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Tumours of the bladder in dyestuff workers have been recorded with increasing frequency during the last 50 years. A high proportion of the recorded cases are attributable to exposure to b-naphthylamine or benzidine; experimental evidence of their carcinogenicity has accumulated during recent years. Hueper and Wolfe (1937), Hueper, Wiley, and Wolfe (1938), and Bonser (1943) have produced malignant bladder tumours in dogs with b-naphthylamine. With benzidine, Spitz, Maguigan, and Dobriner (1950) have obtained tumours of the acoustic gland, liver, and large bowel in rats as well as one bladder tumour in a dog, and we have confirmed their results in rats.

The term "aniline cancer" was applied to this hazard in the dyestuffs industry because a number of the early cases of bladder tumours were associated with the manufacture of aniline (Wendel, 1900; Rehn, 1905; Seyberth, 1907), and with dyestuffs from aniline (Rehn, 1895; Bardenheuer, 1904; Seyberth, 1907; Cesa-Bianchi, 1911; Leuenberger, 1912). Owing to the poor fractionation of crude benzol in these early days "anilines" at that time contained considerable amounts of toluidines and xylidines. It was in fact the presence of p-toluidine as an impurity that enabled Perkin in 1856 to discover mauveine, the first synthetic dyestuff. Recent reports tend to discount aniline as a cause of tumours arising during the last 20 years, although a variety of other aromatic compounds have been suspected (Gehrmann, Foulger, and Fleming, 1949; Goldblatt, 1949; Barsotti and Vigliani, 1949; Müller, 1951; Scott, 1952).

Hergt (personal communication), who has records of a number of early cases in Germany, finds that they were rare in aniline makers but not uncommon in workers making toluidines and xylidines. He believes that these early "aniline tumours" are attributable to toluidines and that their complete absence in men starting work in Germany during the last 40 years is due to improved working conditions, resulting in considerably reduced exposure. The first tumours described by Rehn (1895) were in fuchsine workers, who would be heavily exposed to aniline, toluidines, and xylidines in the course of their work. There is, however, no reliable experimental evidence that either aniline, toluidines, or xylidines are carcinogenic. In an attempt to find an explanation for the early tumours attributed to these bases, a survey was made of the literature relating to the manufacture of aniline in Germany before 1900. This revealed that in 1862 Hofmann isolated a new based, 4-aminodiphenyl (xenylamine), from the distillation residues of "heavy aniline" (*anilin für rot*). Loring Jackson, a collaborator of Hofmann, continued this work, and apart from isolating naphthylamines from similar residues, found therein a monomethyl homologue of aminodiphenyl melting at 46·5°–47·5°C. (acetyl derivative, m.p.114·2°C.) (Loring Jackson, 1875). This latter amine has not been identified but it would not appear to be any of the known isomers.*

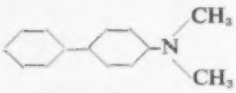
Hofmann (1862) thought that the 4-aminodiphenyl in the residues arose from the cracking of aniline, but Loring Jackson's finding of naphthylamines suggests that these bases were formed by the nitration and reduction of naphthalene, diphenyl, and methyl diphenyl (as well as of toluene and xylenes) all occurring as impurities in the benzene used for making aniline. Kruber (1932) has in fact isolated 2-methyldiphenyl and 3-methyldiphenyl from coal tar and Loring Jackson's amine is possibly a derivative of one of these hydrocarbons. Kruber isolated 4 : 4′-dimethyl and 3 : 4′-

* The known monomethyl homologues of aminodiphenyl are 3-methyl-2-aminodiphenyl, m.p.62–63°C., 4′-methyl-4-aminodiphenyl, m.p.97°C., 3-methyl-4-aminodiphenyl, m.p.41–42°C., or 2′-methyl-4-aminodiphenyl, m.p. not given (acetyl derivative m.p.146–147°C.).

dimethyl-diphenyl at the same time and we see no reason to doubt that other isomers also were present.

As methods of distillation have improved greatly it is not surprising that examination of present-day residues from the distillation of aniline has failed to reveal the presence of naphthylamines, 4-aminodiphenyl, or its homologues.

If the naphthylamine present in Loring Jackson's aniline residues resulted from the nitration and reduction of naphthalene contaminating his benzene, it would be equivalent to commercial a-naphthylamine, which is of low carcinogenic power. We therefore thought it worth while to determine whether 4-aminodiphenyl and its methyl homologues were carcinogenic and, if so, whether they were sufficiently active to have played a significant part in the genesis of early "aniline" tumours. We were encouraged to believe that 4-aminodiphenyl might prove to be carcinogenic by the recent demonstration by Miller, Miller, Sandin, and Brown (1949) that N:N-dimethyl-4-aminodiphenyl produces tumours in the rat and by our own findings and those of Spitz, Maguigan, and Dobriner (loc. cit) that benzidine (4 : 4′-diaminodiphenyl) has a

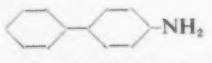


N:N-dimethyl-4-aminodiphenyl

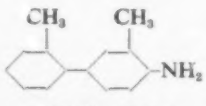
4:4′-diaminodiphenyl (benzidine)

similar action. In selecting a methyl homologue for first test in comparison with 4-aminodiphenyl itself we chose 3 : 2′-dimethyl-4-aminodiphenyl.

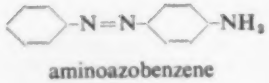
This choice was made because, in the azo series of liver carcinogens, 4-amino-3 : 2′-azotoluene is known to be more active than aminoazobenzene itself. As will be seen below, the disposition of methyl and amino groups in 3 : 2′-dimethyl-4-aminodiphenyl is analogous to that in 4-amino-3:2′-azotoluene.



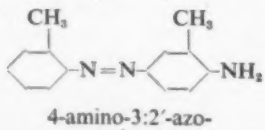
4-aminodiphenyl



3:2′-dimethyl-4-aminodiphenyl



aminoazobenzene



4-amino-3:2′-azotoluene

Preparation of Compounds

4-Aminodiphenyl.—4-Nitrodiphenyl was prepared by the Gomberg reaction (Elks, Haworth, and Hey, 1940) and purified by chromatography. This was reduced with iron and ferrous chloride in aqueous xylene and the final product was fractionated through a gauze packed column (b.p.302°C.) at atmospheric pressure.

3 : 2′-Dimethyl-4-aminodiphenyl.—This compound was prepared by Dr. R. H. Wilson especially for this experiment (Wilson, in press).

Animal Experiments

The animals were albino rats from a closed but random mated colony which has been maintained for some years in these laboratories. They were housed upon sawdust in galvanized wire mesh cages and were allowed to feed at will upon a composite diet in pellet form, with tap water available at all times. The diet was supplied by Scottish Agricultural Industries Ltd., and had the following percentage composition by weight.

Ground barley ..	26·5	Dried skimmed milk ..	13·0
Ground oats	18·5	Meat and bone meal ..	8·6
Bran	18·3	White fish meal ..	4·4
Maize meal	8·6	Dried yeast	1·3
Salt	0·4	"Nuclo"*	0·4

At the start of the experiment the rats were 3 months old and each weighed about 100 g. They were weighed and dosed on a body weight basis five days a week throughout the dosing period. The dosing of individual animals was temporarily stopped if they lost weight and the length of the dosing period varied between groups according to the response observed.

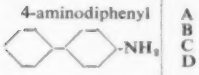
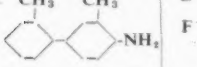
The compounds were dissolved in arachis oil (B.P.) and the solutions given by subcutaneous injection alternately in the right and left pectoral region. Twelve male and 12 female rats were given 4-aminodiphenyl and like numbers arachis oil alone as a control: 24 male rats were treated with 3 : 2′-dimethyl-4-aminodiphenyl. Sub-divisions of the groups receiving the two chemicals were dosed at different levels in order to gain information upon the effect of dosage on response. The details of the dosage are shown in Table 1.

Results

The results given in Table 2 show that a large proportion of the treated animals developed tumours in a variety of sites. Whereas the rats given 3 : 2′-dimethyl-4-aminodiphenyl were all killed by the 259th day, the 4-aminodiphenyl animals survived much longer. All except two of the 23 control rats survived considerably longer than the 260 days. Twelve out of these control animals survived 500 days or over as against 14 in the group given 4-aminodiphenyl. The incidence of tumours at various sites in the different groups will now be compared.

* "Nuclo" is a proprietary vitamin concentrate containing 800 I.U. of vitamin A and 100 B.S.I. units of vitamin D_3 per g.

TABLE 1
DOSAGES OF EXPERIMENTAL RATS

Compound	Group	No. of Rats	Sex	Mean Duration of Dosing (days)	Mean Total Dose (g./kg.)	Mean Total Volume of Oil (ml./kg.)
4-aminodiphenyl 	A	5	Male	376	5·8	280
	B	6	Female	286	4·2	235
	C	6*	Male	291	4·4	236
	D	6	Female	250	3·6	203
3 : 2′-dimethyl-4-aminodiphenyl 	E	12	Male	141	2·8	70
	F	11†	Male	131	3·5	69
Arachis oil ..	G	12	Male	274		331
	H	11‡	Female	296		347

* One died on the 37th day. † One died on the 45th day. ‡ One died on the 30th day.

Tumours at the Site of Injection.—These tumours were observed only in rats which lived more than 400 days. They occurred in five control rats and in four of those given 4-aminodiphenyl. It will be seen that none developed in the animals receiving 3 : 2′-dimethyl-4-aminodiphenyl, all of whom died or were killed within 260 days. The occurrence of these tumours in both the control group given arachis oil and those given 4-aminodiphenyl in arachis oil leads us to believe that they were due to the oil.

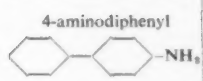
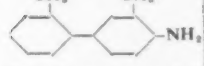
Compound	Times of Appearance of Tumours (days)
4–aminodiphenyl	418, **498**, **500**, 502
Controls (arachis oil)	**421**, **537**, **544**, **575**, **576**
3 : 2′–dimethyl–4–amino-diphenyl	Nil

Sarcomata in bold type, the remaining two being fibromata.

The local tumours were all at least 2 cm. in diameter. Two tumours consisted of well-differentiated coarse connective tissue with few cells and were considered to be fibromata. The remainder had the histological appearance of sarcomata of either spindle cell or mixed cell type. No metastases had occurred, but in the sections examined local infiltration of surrounding tissue was present in one tumour of the arachis oil group and in one where 4-aminodiphenyl had been given.

Mammary Tumours.—These are not unknown in untreated animals of our stock, although none appeared in the control group of 12 males and 11 females in this experiment given arachis oil alone. The appearance of two adenocarcinomata

TABLE 2
INCIDENCE* AND CLASSIFICATION OF TUMOURS

Compound	Group	Number of animals	Sex	Benign hepatoma	Benign cholangioma	Reticulosis	Sarcoma of liver	Intestinal tumours	Mammary tumours	Fibroma at injection site	Sarcoma at injection site	Tumours of salivary glands	Tumours of acoustic sebaceous glands	Papilloma of bladder	Adenoma of kidney	Carcinoma of uterus	Subcutaneous carcinoma of unknown origin	Number of normal animals	Times (days) when Normal Animals Died or Were Killed
4-aminodiphenyl 	A	5	Male	—	1	—	1	1	—	—	—	—	—	—	—	—	—	2	(346 and 537)
	B	6	Female	—	—	—	—	2	1	—	1	—	—	—	—	1	1	2	(364 and 398)
	C	6	Male	1	—	1	—	3	—	2	—	—	—	—	1	—	—	—	
	D	6	Female	—	—	—	—	1	2	—	1	—	—	—	—	1	—	2	(105 and 523)
3 : 2′-dimethyl-4-aminodiphenyl 	E	12	Male	—	—	—	—	12	1	—	—	4	4	—	—	—	—	—	
	F	11	Male	—	—	—	—	9	—	—	—	1	—	1	—	—	—	2	(69 and 119
Controls	G	12	Male	—	—	—	—	—	—	—	1	—	—	—	—	—	—	11	(73, 253, 322, 329, 383, 435, 572, 572, 572, 575 and 575
	H	11	Female	—	—	—	—	1	—	—	4	—	—	—	—	—	—	7	(262, 434, 492, 501, 575, 582 and 582)

* The figures refer to the number of *rats* bearing the types of tumour listed.

and one adenofibroma with areas of exuberant growth amongst 12 females given 4-aminodiphenyl and one adenocarcinoma in a male given 3 : 2′-dimethyl-4-aminodiphenyl must be regarded as abnormal.

Tumours Involving the Salivary Glands.—These tumours were observed only in the group given 3 : 2′-dimethyl-4-aminodiphenyl. They occurred at 193, 193, 216, 224, and 252 days respectively and were sarcomata infiltrating and strangling the salivary glands. They were not associated with oil collected at the injection site as were the fibromata and sarcomata observed at a much later date in the other two groups. Tumours involving these glands have been described by Heimann and Meisel (1946) in animals given acetylaminofluorene by mouth.

Tumours of the Acoustic Sebaceous Gland.—Tumours of this gland occurred in four rats given 3 : 2′-dimethyl-4-aminodiphenyl. They were first observed at 153, 156, 173, and 176 days respectively. In one rat a squamous cell carcinoma developed, whereas bilateral tumours of the same type occurred in another (Fig. 1). A third developed a sebaceous cell carcinoma, and the fourth gross cystic enlargement of the gland with thick irregular squamous stratified epithelium lining the cysts.

Lesions of the Liver.—No liver tumours were observed in the control group but some of these older animals did show a mild proliferation of the bile ducts. In the 4-aminodiphenyl group one animal developed a large benign cholangiomatous cyst with no invasion of the liver parenchyma. Another developed a hepatoma whilst a third had an extensive sarcoma in the liver which infiltrated the pancreas as well. No primary sarcoma was found elsewhere in this rat. Yet another animal in this group developed a reticulosis, with gross infiltration of the whole liver. Cells were escaping into the circulation and were found in the lung vessels. They were not, however, forming secondary infiltrations.

No gross changes were observed in the livers of the animals given 3 : 2′-dimethyl-4-aminodiphenyl.

Tumours of the Intestinal Tract.—One tumour of the caecum was observed in a control rat killed at 570 days. This lesion was raised on the internal surface of the bowel, and atypical disorganized glands penetrated the muscularis mucosae. No other tumours of the intestine occurred in the control group.

Tumours of the intestines occurred in all but two of the animals treated with 3 : 2′-dimethyl-4-aminodiphenyl. In almost every case they were multiple and appeared along the whole length

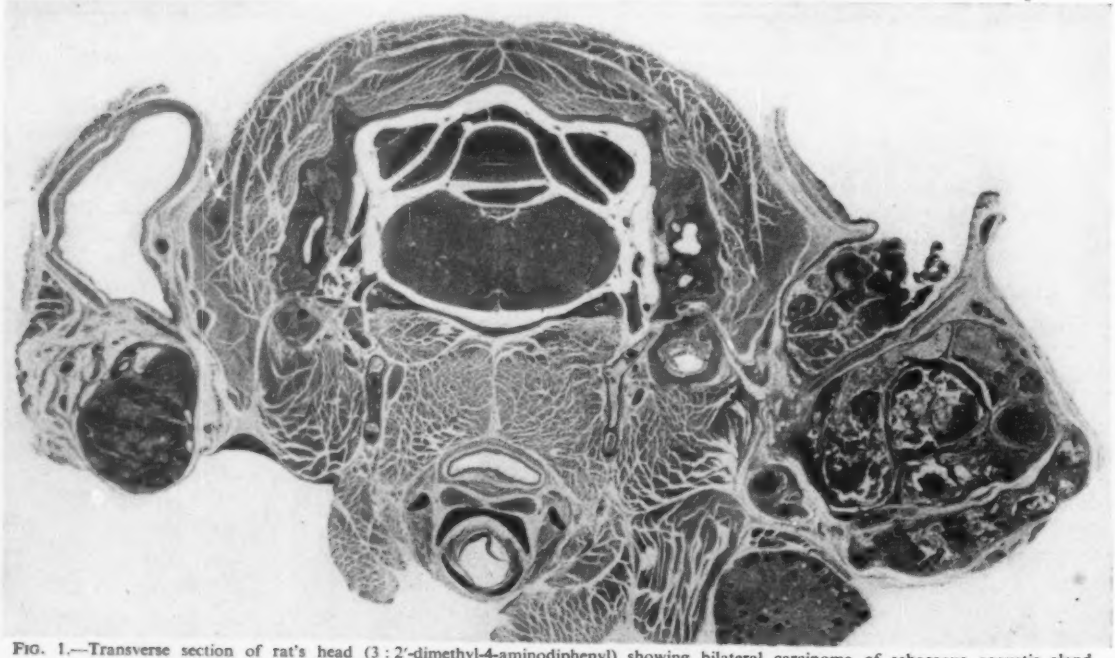


FIG. 1.—Transverse section of rat's head (3 : 2′-dimethyl-4-aminodiphenyl) showing bilateral carcinoma of sebaceous acoustic gland. Note invasion of external auditory meatus on one side. (×4)

FIG. 2.—Abdominal contents of rat (3 : 2′-dimethyl-4-aminodiphenyl) showing multiple lesions of the large and small intestines.

of the large and small intestine (Fig. 2). In only one rat did a tumour occur in the small intestine when there were none in the large bowel.

In the group given 4-aminodiphenyl tumours occurred only in the large intestine. They were found in seven out of the 14 rats which survived over 500 days.

In the 3 : 2′-dimethyl-4-aminodiphenyl series the animals were killed because of haemorrhage, diarrhoea, or intestinal obstruction often associated with intussusception. Thus the relative times of discovery of the tumours in the three groups have some significance, although the majority of 4-aminodiphenyl tumours occurred in the caecum where obstruction was less likely to occur. No tumours of the forestomach were observed in any of the animals.

Compound	Times of Appearance of Intestinal Tumours (days)
3 : 2′–dimethyl–4–aminodiphenyl	80, 104, 138, 146, 149, 160, 167, 189, 190, 191, 191, 193, 195, 205, 209, 216, 248, 259, 259, 259 and 259
4–aminodiphenyl	570, 570, 570, 570, 576, 577 and 577
Controls	570

It is clear that all the tumours in the group given 3 : 2′-dimethyl-4-aminodiphenyl developed very much earlier than those in the other groups. This fact, coupled with the incidence of tumours of the small intestine in this group, is taken as an indication of the greater carcinogenic activity of 3 : 2′-dimethyl-4-aminodiphenyl as against 4-aminodiphenyl itself.

The tumours of the large intestine varied considerably in type and extent. Those involving several centimetres of the intestine showed generalized thickening of the gut wall associated with multiple hard nodules on the peritoneal surface. The early tumours tended to be of this type and sometimes the mucosal surface was ulcerated whereas later lesions appeared as either sessile reddened adenomata or pedunculated tumours. The latter were particularly prone to be carried down the lumen with resultant intussusception.

The histological picture of tumours of the large intestine may be divided into the following classes for descriptive purposes but, in fact, a variety of intermediate lesions was observed. The smallest tumours were characterized by a thickening of the submucosa associated with disorganization and hypertrophy of the glands (Fig. 3). The

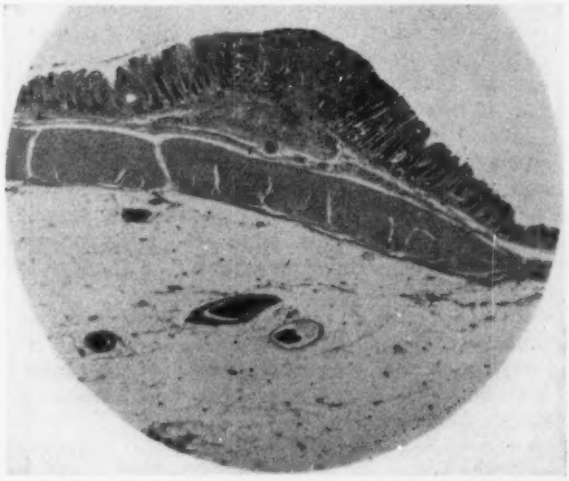


FIG. 3.—Early lesion of large intestine of rat (4-aminodiphenyl). (×12)

larger, sessile adenomatous type showed an increase of this process and thickening of the bowel wall with or without breaching of the muscularis mucosae, which was often drawn up on either side of the tumour. The glands extended down into the submucosa, sometimes with cyst formation, infiltration with leucocytes and eosinophils, and associated thickening of the peritoneum. Alternatively, the tumour took on a pedunculated form with considerable hypertrophy of the glands, the muscularis mucosa being drawn up into the

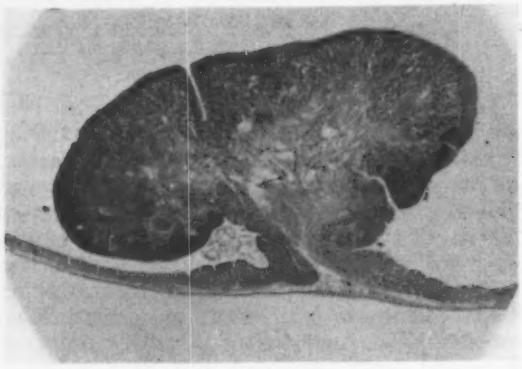


FIG. 4.—Adenomatous polyp of large intestine of rat (4-aminodiphenyl). (×6)

connective tissue core (Fig. 4). In the most marked lesions, disorganized and atypical glands, usually hyperchromatic and containing a diminished number of goblet cells, repeatedly penetrated the submucosa and invaded or penetrated the muscularis externa forming cystic projections on the bowel surface covered with thickened peritoneum (Fig. 5). In these latter cases the circular muscle often showed local thickening and loss of polarity as described by Stewart and Lorenz (1946–47).

The tumours of the small bowel followed a similar pattern with local thickening of the bowel and protrusion of hyperplastic glandular tissue into the lumen. In some other cases the muscle layers were breached and cystic projections covered with thickened peritoneum appeared on the surface of the small bowel.

In our lesions of the large and small intestines there was no infiltration of surrounding tissues as occurred in the adenocarcinomata of the colon described by Spitz, Maguigan, and Dobriner (1950). Our material shows a marked similarity to the lesions of the bowel of mice following oral administration of 20-methylcholanthrene and 1, 2, 5, 6-dibenz-anthracene described by Stewart and Lorenz (1946–47). Indeed, following Stewart's classification, our most advanced tumours could be considered as adenocarcinomata. On the other hand, Lushbaugh and Hackett (1948–49), who obtained infiltrating adenomatous lesions of the colon in rats given lubricating oil by mouth, inclined towards a diagnosis of reactive hyperplasia for lesions which again closely resembled those described here.

Further work which is proceeding on the characterization of these intestinal lesions will be described in a later communication.

Discussion

Our results demonstrate that 4-aminodiphenyl produces tumours in the rat at various sites, including the intestinal tract. Benzidine also produces tumours of the intestine in the rat and causes bladder tumours in man. By analogy 4-aminodiphenyl also may be capable of causing human bladder tumours. In view of this possibility, we suggest that the manufacture of 4-aminodiphenyl for industrial use should be carried out only with the most stringent precautions.

Despite our belief that 4-aminodiphenyl and commercial a-naphthylamine are capable of producing bladder tumours in man, we do not believe that the small amounts of these high-boiling amines present in distillation residues could have accounted fully for the early tumours amongst "aniline" makers. It appears to be even less likely that significant quantities of these amines would have been present in the distillate used subsequently for dyestuff manufacture. Similar conclusions can be reached regarding compounds as active as 3 : 2′-dimethyl-4-aminodiphenyl. Nevertheless our results are of intrinsic interest and have led us to develop a hypothesis concerning the carcinogenic action of aromatic amines.

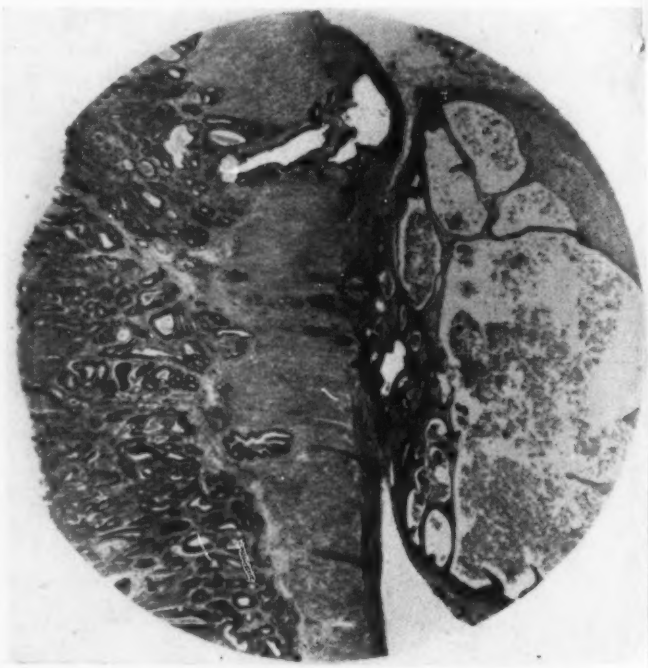
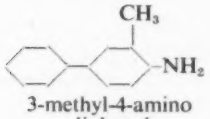


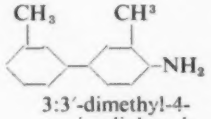
FIG. 5.—Adenocarcinoma of the large intestine of rat (3 : 2′-dimethyl-4-aminodiphenyl) showing glands infiltrating through muscularis externa and forming cyst-like projections under peritoneal surface. (×23).

Our experiments show that 3 : 2′-dimethyl-4-aminodiphenyl produces distant tumours earlier and more frequently than 4-aminodiphenyl itself. The tumours obtained by Miller and others (1949) with N : N-dimethyl-4-aminodiphenyl also appeared earlier and in a higher proportion of animals than we observed with 4-aminodiphenyl. It will be recalled that 4-amino-3 : 2′-azotoluene and N : N-dimethylaminoazobenzene are much more active liver carcinogens than aminoazobenzene itself. It thus appears that 3 : 2′-dimethyl or N : N-dimethyl substitution augments carcinogenic activity in both the 4-aminodiphenyl and aminoazobenzene series.

In experiments as yet uncompleted tumours of the intestine have appeared at 106 days with 3 : 3′-dimethyl-4-aminodiphenyl and at 120 days with 3-methyl-4-aminodiphenyl. These times correspond very closely with the earliest appearance of tumours in our animals treated with 3 : 2′-dimethyl-4-aminodiphenyl. These latter results lead us to believe that it is the methyl group ortho to the amino rather than the methyl group in other ring positions which is responsible for the increase in activity in this series.



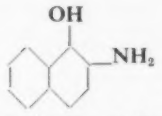
3-methyl-4-amino diphenyl



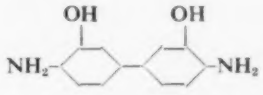
3:3′-dimethyl-4-aminodiphenyl

Since the incidence of tumours at the site of injection of 4-aminodiphenyl and its homologues in no cases exceeded that in the controls, it appears that the compounds do not exert a direct and local carcinogenic action, such as is observed with polycyclic hydrocarbons of the type of dibenzanthracene or methyl cholanthrene. We conclude that the effective carcinogen is in all cases a metabolite of the amine given and that the appearance of tumours throughout the intestinal tract (excluding the forestomach) in the rat suggests that these metabolites are excreted in this species in the bile.

Recently published work upon two aromatic amines which similarly produce only distant tumours suggests that the effective metabolite may be an ortho-hydroxy amine. Engel (1920) showed that b-naphthylamine was metabolized by the dog to 2-amino-1-naphthol, and Hueper (1938) obtained evidence suggestive of carcinogenicity by injecting the latter compound intraperitoneally into mice. Bonser, Clayson, and Jull (1951) have shown that 2-amino-1-naphthol has a local carcinogenic action on the bladder epithelium of mice, and have obtained (personal communication) further evidence that 2-amino-1-naphthol is locally carcinogenic although control tests were negative. Further, Clayson has been preparing o-hydroxy amines related to other carcinogenic aromatic amines for bioolgical testing.



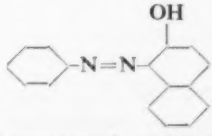
2-amino-1-naphthol



3:3′-dihydroxybenzidine

Adler (1908) showed that benzidine was metabolized by the rabbit to a dihydroxy derivative of melting point 130–138°C. Baker (1950) isolated a compound from human urine subsequently identified as 3 : 3′-dihydroxybenzidine and has shown (personal communication) that when fed to rats it rapidly produces intestinal tumours. These intestinal tumours appeared considerably earlier than any obtained by Spitz, Maguigan, and Dobriner (1950) or by ourselves by injecting benzidine itself subcutaneously. This and the fact that Baker obtained tumours of the forestomach suggests that 3 : 3′-dihydroxybenzidine may be the effective metabolite.

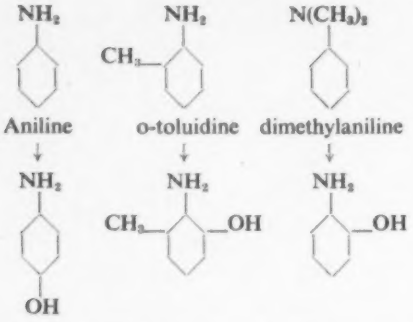
A further example of an o-hydroxy amine acting as a carcinogen may be deduced from the finding of Kirby and Peacock (1949) that benzeneazo-b-naphthol is a liver carcinogen in the mouse. Reductive fission of the azo linkage is well known to occur in this animal, and would yield 1-amino-2-naphthol in this case.



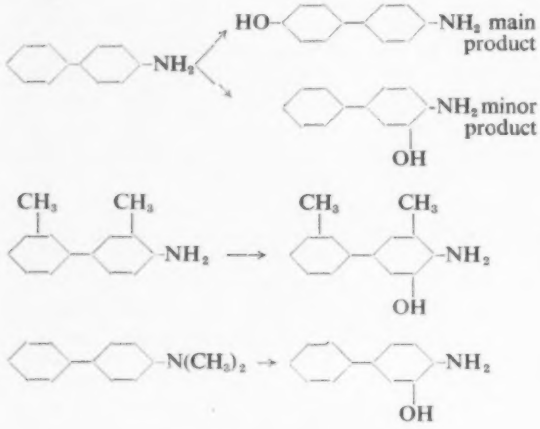
benzeneazo-b-naphthol

Although direct evidence is not as yet available regarding the metabolism of compounds of the 4-aminodiphenyl series, results obtained with aniline and its derivatives in the dog indicate that o-methyl or N : N-dimethyl substitution results in the "steering" of metabolic hydroxylation from the para into the ortho position relative to the amino group.

Thus aniline is mainly metabolized to para-aminophenol by the dog, whereas o-toluidine is excreted largely as o-amino-m-cresol. This picture is made complete by the fact that the dog demethylates dimethylaniline and excretes o-aminophenol (Williams, 1947).



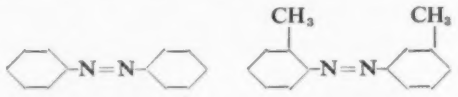
We suggest that the metabolism of the corresponding derivatives in the 4-aminodiphenyl series follows the same pattern as shown below, although it is again pointed out that we have no direct evidence of this.



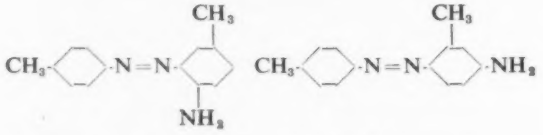
If in this case also the o-hydroxyamines are the effective carcinogenic metabolites, this would explain the increased carcinogenic activity of the methyl substituted amines as compared with 4-aminodiphenyl itself.

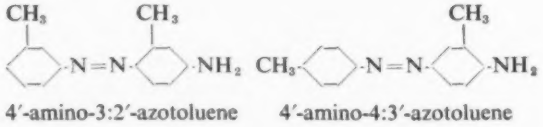
If the suggestion that o-hydroxyamines are direct-acting carcinogens is valid for such simple compounds as o-aminophenol, we should expect o-toluidine and dimethylaniline, but not aniline, to produce bladder tumours in the dog. Gehrmann and others (1949) in fact state that they have been unable to produce tumours in the dog with aniline. In order to obtain evidence to substantiate these hypotheses, experiments have been started in which o-toluidine and dimethylaniline will be given for long periods in high dosage to dogs.

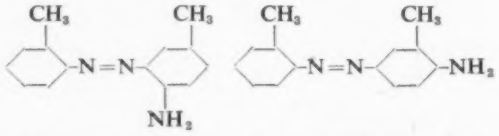
Indirect support for the suggestions, first, that simple o-hydroxyamines are direct acting carcinogens and, secondly, that the metabolic yield of them from an amine is increased by o-methylation or N : N-dimethylation, may be deduced from the observation that 2 : 3′-azotoluene yields bladder papillomata in rats (Otsuka and Nagao, 1936) whereas azobenzene itself is inactive as a carcinogen (Spitz, Maguigan, and Dobriner, 1950). Reductive fission of the azo linkage would yield o- and m-toluidine in case of 2 : 3′-azotoluene, whereas azobenzene would only yield aniline.



The relationship between structure and carcinogenic activity in other azo compounds can be explained in the same way, although clearly the reducibility of the azo linkage will exert a considerable influence on the result. Thus, of the six following isomers of aminoazotoluene fed by Crabtree (1949) to mice, only one, namely 2′-amino-4 : 5′-azotoluene, failed to show any activity, and this is the only one of these which on reductive fission would not form an o-methyl amine. Furthermore, activity was highest in 4′-amino-2 : 3′-azotoluene which would divide to form two molecules each containing an o-methyl amine.







Finally this hypothesis provides a theoretical explanation for Hergt's view that the early "aniline tumours" amongst dyestuffs workers were due to o-toluidine, absorbed by workmen in considerable

quantities during the early days of the chemical industry. The fact that such tumours, unassociated with contact with b-naphthylamine or benzidine, are very rare today suggests that modern conditions have removed the heavy contamination necessary for the production of such tumours in man.

Summary

4-Aminodiphenyl has been shown to be carcinogenic in the rat and it is suggested that it may be capable of causing bladder tumours in man. The presence of this base, together with naphthylamines, in the distillation residues of "heavy aniline" is thought to have been partly responsible for early "aniline cancers".

3 : 2′-Dimethyl-4-aminodiphenyl has been shown to be more potent than 4-aminodiphenyl itself as a carcinogen in the rat. It has been suggested that in each case the effective carcinogen is an o-hydroxyamine, formed in the body by metabolic hydroxylation, and that the higher activity of the 3 : 2′-dimethyl homologue of 4-aminodiphenyl is due to the preferential direction of the hydroxy group into the position ortho to the amino group under the influence of the 3-methyl group.

It is suggested that o-hydroxy amines as simple as o-aminophenol may be direct-acting carcinogens. On this basis it is possible to explain the relationship between structure and the capacity of azo compounds to induce tumours of the liver in experimental animals. On this basis also it appears likely that o-toluidine may be capable of producing bladder tumours in man and the dog.

We wish to thank Dr. A. Hergt for unpublished information and Dr. R. H. Wilson and Dr. E. G. Parry for preparing the chemicals. We are also grateful to Miss B. Lowe and Miss P. Nutty for technical help.

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Since this paper was submitted Bonser, Clayson, and Jull have shown that commercial 1-amino-2-naphthol is also a local carcinogen.

Brit. J. industr. Med., 1952, **9**, 264.

SICKNESS ABSENCE RECORDING IN INDUSTRY

BY

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Time lost in industry may be due to normal and extraordinary leave, certified and uncertified sickness, accidents, leave without permission, and lateness. Clearly the " unexpected " absence, that is leave due to sickness and accident, absence without permission and lateness, is the most upsetting to industry since normal and extraordinary leave are accepted, and, in the main, foreseen. Furthermore, time lost by a worker sometimes means additional time lost by the group in which he or she works.

Of the " unexpected " absences sickness is the greater part, and there is evidence that certified sickness is much less influenced by socio-economic factors and legislation than are other unexpected absences. In any industrial concern there will always be some sickness unrelated to the type of work, or to personal relationships, pay, promotion, bonuses, feelings of importance or lack of importance, and good or bad management. Where, however, there are adverse environmental and social factors of this kind there may be an increased sickness absence, although this is not necessarily so, but there is always a still greater increase in absences from other causes. For instance, Behrend (1951) found in 46 factories in 1948 that " there appears to be no close connection between the level of the other reasons absence rate and the certified sickness absence rate ", and, similarly, that there was only a negligible difference between the sickness absence rate in the 10 factories with the highest " other reasons " rate and that in the 10 with the lowest rates for other reasons. (" Other reasons " includes authorized leave.)

New cases of sickness absence are more commonly recorded as starting on Mondays and less commonly on Fridays, with a gradation between the two. Clearly Monday shows a peak because illness may have started on the preceding Saturday or Sunday, but as Behrend points out, the average daily certified sickness rate should be the same on every day of the week, with the exception that most return to work on a Monday and least on a Friday. In a company employing 370 men studied for nine months she found the average weekly certified sickness absence rates to be 3·0% on Monday and 3·2% on Friday. The highest level was 3·3% on Wednesday. There was therefore little difference throughout the week. Similarly in the coal-mines Behrend showed that the sickness rate was lowest on Monday and highest on Saturday, in contrast to the voluntary absence rate which was highest on Monday and Saturday and lowest on Friday.

These findings, taken in conjunction with Behrend's other demonstration of a lack of correlation between certified sickness absence and "other reasons " absence, suggest that a separate study of sickness absence is worth while because in the main it is caused by true morbidity in the working population. Until there are adequate statistics of this kind in all sections of industry, it is impossible to apply preventive measures for the benefit of the work people and of the firms employing them. Adequately reliable data may be obtained from the statutory medical certificate, and small concerns should find it to their advantage to record and analyse them. Raffle (1950) in a large working group showed that in sickness and accident of long duration 80% of the diagnoses given on general practitioners' medical certificates fell into the same broad groups (of 21 headings) as the diagnosis subsequently made by the medical officers of the industry.

Employers often want to know how the sickness experience of persons employed by them compares with a similar labour force in other factories. If their own rate is high then a detailed analysis may point a remedy and allow measures to prevent ill-health to be taken. The comparisons required, even if the necessary data were available, are not quite as simple as they may appear to be. Sickness absence may be influenced by light or heavy work ; for example, a person with a chronic disability may be able to do a light job but not a heavy one and

the same will often apply to a temporary disability. It is influenced by age, an important factor in an ageing population, and it is influenced by sex. Spratling and Lloyd (1951) point out that " the normal amount of sickness absence for men in the late fifties is about two or three times as much as it is in the twenties or early thirties ", while that " of women is normally more than that of men and that of unmarried women more than that of unmarried men ". Various socio-economic factors which enter into everyone's life will also exert some influence on the amount of sickness experienced, though far less, it seems, on certified sickness than on other unexpected absences. It will be influenced by medical selection applied at entry, and, artificially, by the length of time a person is kept on the books.

Thus, comparisons of sickness absence between factories or between industries must be made with discretion. Within the same factories, or group of factories, with comparable work, inter-departmental or occupational comparisons may be made much more readily and with profit. Such comparisons may reveal unsuspected levels of sickness absence from particular causes.

For such purposes the first and essential step is the development of a simple and uniform method of recording sickness. An investigation, made in 1950, of the medical facilities of a large group of factories in London, each of which employed up to 500 persons, revealed that only one in six had the part-time services of a doctor and few had any nursing service. If this picture is true of industry as a whole the great majority of factories lack any means by which disease or accident would normally be recorded in such a way as to reveal any excessive number or unusual nature of such occurrences. It should also be remembered, as Table 1 shows, that British industry still consists largely of relatively small units.

Observation of the smaller units of under 50 work people should hardly call for any detailed form of sickness absence recording ; in the largest, 1,000 employees or more, usually some system has already become a necessity. The simple system of recording to which this present study is devoted is, therefore, intended to be applicable and a guide to factories from 50 to 1,000 strong. Table 1 shows, assuming that all the entries are autonomous units, that these number some 23,000, or one in eight of employers, and that they employ within their factories some 4,000,000 operatives or over half of the total labour force of factories.

While the system of recording set out below is most easily used in factories, the same principles can be, and have been, successfully used in other sections of industry. They may, too, it is hoped, be of use to medical officers acting as consultants to any occupational group.

TABLE 1

SIZE OF FACTORIES AT DECEMBER, 1949

Size of Firm (Nos. Employed)	No. of Factory Units	%	Cumulative %	No. of Employees (000's)	%	Cumulative %
1–10	132,338*	70·6	—	537*	7·2	—
11–24	17,114	9·1	79·7	289	3·9	11·1
25–49	14,171	7·6	87·3	498	6·7	17·8
50–99	10,138	5·4	92·7	710	9·5	27·3
100–249	8,134	4·3	97·0	1,265	16·9	44·2
200–499	3,190	1·7	98·7	1,103	14·7	58·9
500–999	1,439	0·8	99·5	984	13·1	72·0
1,000–1,999	601	0·3	99·8	822	11·0	83·0
2,000 and over	342	0·2	100·0	1,277	17·0	100·0
Total ..	187,467	100·0	—	7,485	100·0	—

Data obtained from the *Ministry of Labour Gazette* for June, 1950.

* Firms employing 10 employees or less are not now required to make returns. These figures were obtained from the Census of Production Statistics of 1935 quoted in the *Ministry of Labour Gazette* for April, 1948. At this time the number of factories employing 10 or less persons was 73% of the total number of factories, so that little error will be made by their use.

Short and Long Absences

It has been generally agreed that a division should be made between short absences and those of greater length. The division is usually made at four days, absences of under this time (one, two, or three days) being termed " short " and those of four days or more being termed " long " (Medical Research Council, 1944; Spratling and Lloyd, 1951). Though long absences are in many ways the more important, there is no doubt that short absences should also be fully recorded and studied, since they invariably form a large proportion of the total sickness absence (sometimes as much as one half) and have quite a different pattern of causes. Many will be unsupported by a medical certificate, and, as Spratling and Lloyd suggest, it may well be that " social, economic, and administrative factors exert a greater influence than the purely medical " on them. To ignore these relatively numerous terms of short sickness is, however, to lose information on much general, though minor, morbidity, and as part of these absences are certified it is arguable that short absence data are not appreciably less reliable than those of long.

To ensure comparability in the sickness absence rates of different concerns it is also necessary to place some upper limit on an absence. Otherwise, as already pointed out, the rates will be artificially swollen or shrunken by the length of time a sick person is kept on the books. The Medical Research Council and Spratling and Lloyd place this limit

at six months (or 182 days) and suggest that sickness absence in excess of this should be analysed separately. This division has been adopted here, though it may be noted that in small firms the amount of data relating to absences of over six months is unlikely to be sufficient to repay analysis.

Method of Recording Sickness

The larger firm which possesses a personnel or medical department may use either of these services for its sickness absence recording. Of the two, the personnel department is generally the more suitable unless the medical officer is whole-time, for it is not unusual to find that there is lack of liaison in questions of absence in these departments where medical advice is not always available, consequently a medical officer may keep his own statistics of consultations while the personnel officer keeps records from observations of medical certificates.

In a small firm lacking a personnel officer a suitable department must be found within the existing framework without, if possible, increasing the staff. Fortunately the " pay point " is readily suitable for this. In the wages department it is necessary to have day-to-day information about employees solely for the purposes of calculating pay. If a worker is absent he is recorded as such and the recording of why the individual is away is merely a simple extension of existing services. Also the wages department know the numbers of persons currently employed and the hours which they work in every branch of the organization.

In concerns with sub-units which make returns to a central wages department sickness absence is best collected by the manager at the head of the sub-unit. The responsible person should include on the return not only the numbers of days absent by any worker but also the reason for absence, which, for sickness or accident, will include the diagnosis on the medical certificate. This record relates to both an individual and to a group and is permanent. The individual items are transferred to the employee's personal documents, the diagnosis being coded, i.e. allocated to a group of similar illnesses, by a clerk centrally unless the sub-unit undertakes to code. The form may be designed to fit the particular industry, but one used by the London Transport Executive has proved of great value, and is here reproduced, slightly modified, as Form A. (A small concern could, of course, record directly on to the individual's documents without using this form at all.)

FORM A

CONFIDENTIAL

Concern : Acme Toy Co. *Dept. : Mechanical* *Section : Assembly*

Week ended : 29th March, 1952

Average No. Employed : 83

PERSONS ABSENT THROUGH SICKNESS OR ACCIDENT

Serial and Clock Number		Name and Initials of Persons Absent	Days Absent at Start of Week	Date and Cumulative Number of Days Absent							Reason for Absence*	Diagnosis (in case of accidents† certify on or off duty)	Diagnostic Group		Central Use	
				Sun. 23rd	Mon 24th	Tue. 25th	Wed. 26th	Thu. 27th	Fri. 28th	Sat. 29th					No. of Rest Days	Total Days Absent by End of Current Week
		Males														
1	1234	Brown, A. J.	37	R‡	38	39	40	41	42	43	C	Pleurisy	5	519	7	50
2	1235	Jones, K. L. M.	—	—	—	—	1	2	3	4	C	Nervous debility	2	318	—	4
3	etc.															
		Females														
1	2173	Smith, Mrs. Phyllis	11	R	12	13	14	15	—	—	C	Concussion A.F.	11	852	2	17
2	5021	Robinson, Miss Mary	—	—	—	—	1	2	—	—	NC	Headache	2	791	—	2
3	etc.															

Checked :

Coded :

Entered on personal documents :

‡ Rest Day.

† Accidents : AW = Accident at work.

AF = Accident not at work.

* Reason for absence : C : Sickness or accident supported by medical certificate.

NC : Sickness or accident not supported by medical certificate.

(Signed) A. Branch-Manager.

The essential requirements for the form are: (1) a heading showing the name of the firm, department, unit, etc.; (2) the date(s) of the period under review; (3) space for entries for each individual absent (*a*) clock number, (*b*) name, initials and, for women, whether married or single (including widowed), (*c*) date of first and last day of absence and total number of days absent up to date, (*d*) reason for absence; (4) average number of persons employed during the period under review; (5) the form should be marked "confidential" and have a space for signature by the person responsible for filling it in.

In practice the form shown is perforated at the left-hand margin and is made up in book-form. The second sheet is a carbon copy of the original and is retained by the sender. Rest days are marked with an R and are not included in the cumulative totals of days absent from work, but, as they are important from the medical aspect, they are recorded by the clerk centrally in the penultimate right-hand column of the form, and added to the number of working days absent to give the "total days absent" in the final column.

On the form Mr. A. J. Brown had, at the beginning of the week, already been absent continuously for 37 working days and he has a further week's absence, making a total of 43 working days which will be inserted at the beginning of next week's return, i.e. in the column headed "days absent at start of week". This relates to an unbroken period of absence. Any return to work for even a day brings that period of sickness to an end. During this illness Brown has been ill on seven Sundays, so that his "total days absent" is now 50 days. Unless Mr. K. L. M. Jones returns to work on Monday he will start with four days absence at the beginning of the following week and he will also have lost one rest day by this time.

Women's entries are kept separately. (This is always desirable to avoid confusion but it is not essential where numbers are very small.) Mrs. P. Smith suffered concussion due to an accident away from work, and is therefore recorded as an accident off duty. All except Miss Robinson produced medical certificates, and the diagnosis on the certificate or the employee's statement is entered in the appropriate column. Since the names, etc., of all who were away on the last working day of this week will be again inserted in the first three columns of the following week, unless they return on the Monday, those who have been longest away will automatically appear at the head of the form, and thus a daily tally is kept of the numbers of days any individual is absent. This is of great use when a member is drawing sick benefit over a limited period and for sick clubs and visiting schemes which need a day-to-day knowledge of each worker.

Saturday, when worked, is counted as a whole day (and, of course, if not worked as R). Where the working population is varying the average number employed is obtained by adding the numbers who should be present (this includes the absentees) on the Monday to those who should be present on the Saturday (or Friday in a five-day week) and dividing by 2. Where the population is steady, the number who should be present at the beginning of the week is all that is necessary.

The Individual Records

Form A is the group record. For each worker it is desirable to have two records. One is the master or main record, the other the temporary or annual record.

The Master Record.—The master record is a card which will last the worker throughout his employment. The non-changing, or only rarely changing, information about the individual is entered on it. Two record cards may appear extravagant but they are in fact deliberately suggested in order, first to avoid the waste of paper which occurs where a single document is used for all recording and may have to be renewed several times a year, and secondly to save the clerk's time in constantly transferring the same information (date of birth, etc.) from card to card annually, or when the card is full. Since all industrial concerns need a master record of certain details of their work people, and as the amount will vary with the firm, no attempt has been made to design such a card, but the suggestions made here might be added, where suitable, to existing records or incorporated in subsequent printings.

The master card consists of three parts: (1) means of identification of the employee; (2) unchanging (or infrequently changing) information essential for employment purposes; (3) other information for permanent record.

Under (1) are recorded the name and initials (and marital status for women), year of birth, and clock number. Sex is usually differentiated by the colour of the cards. Under (2) are recorded the employee's address (with space for two changes), date of entry to the firm, details of previous jobs and qualifications for the present one, grading, present occupation, and department. Sufficient space is left for promotions and other changes in status or work. Further spaces are required for recording whether the employee is a young person or a

at six months (or 182 days) and suggest that sickness absence in excess of this should be analysed separately. This division has been adopted here, though it may be noted that in small firms the amount of data relating to absences of over six months is unlikely to be sufficient to repay analysis.

Method of Recording Sickness

The larger firm which possesses a personnel or medical department may use either of these services for its sickness absence recording. Of the two, the personnel department is generally the more suitable unless the medical officer is whole-time, for it is not unusual to find that there is lack of liaison in questions of absence in these departments where medical advice is not always available, consequently a medical officer may keep his own statistics of consultations while the personnel officer keeps records from observations of medical certificates.

In a small firm lacking a personnel officer a suitable department must be found within the existing framework without, if possible, increasing the staff. Fortunately the "pay point" is readily suitable for this. In the wages department it is necessary to have day-to-day information about employees solely for the purposes of calculating pay. If a worker is absent he is recorded as such and the recording of why the individual is away is merely a simple extension of existing services. Also the wages department know the numbers of persons currently employed and the hours which they work in every branch of the organization.

In concerns with sub-units which make returns to a central wages department sickness absence is best collected by the manager at the head of the sub-unit. The responsible person should include on the return not only the numbers of days absent by any worker but also the reason for absence, which, for sickness or accident, will include the diagnosis on the medical certificate. This record relates to both an individual and to a group and is permanent. The individual items are transferred to the employee's personal documents, the diagnosis being coded, i.e. allocated to a group of similar illnesses, by a clerk centrally unless the sub-unit undertakes to code. The form may be designed to fit the particular industry, but one used by the London Transport Executive has proved of great value, and is here reproduced, slightly modified, as Form A. (A small concern could, of course, record directly on to the individual's documents without using this form at all.)

FORM A

CONFIDENTIAL

Concern : Acme Toy Co. *Dept. : Mechanical* *Section : Assembly*

Week ended : 29th March, 1952

Average No. Employed : 83

PERSONS ABSENT THROUGH SICKNESS OR ACCIDENT

Serial and Clock Number		Name and Initials of Persons Absent	Days Absent at Start of Week	Date and Cumulative Number of Days Absent							Reason for Absence*	Diagnosis (in case of accidents† certify on or off duty)	Diagnostic Group		Central Use	
				Sun. 23rd	Mon 24th	Tue. 25th	Wed. 26th	Thu. 27th	Fri. 28th	Sat. 29th					No. of Rest Days	Total Days Absent by End of Current Week
		Males														
1	1234	Brown, A. J.	37	R‡	38	39	40	41	42	43	C	Pleurisy	5	519	7	50
2	1235	Jones, K. L. M.	—	—	—	—	1	2	3	4	C	Nervous debility	2	318	—	4
3	etc.															
.																
.																
.																
		Females														
1	2173	Smith, Mrs. Phyllis	11	R	12	13	14	15	—	—	C	Concussion A.F.	11	852	2	17
2	5021	Robinson, Miss Mary	—	—	—	—	1	2	—	—	NC	Headache	2	791	—	2
3	etc.															

Checked :

Coded :

Entered on personal documents :

‡ Rest Day.

† Accidents : AW = Accident at work.

AF = Accident not at work.

* Reason for absence : C : Sickness or accident supported by medical certificate.

NC : Sickness or accident not supported by medical certificate.

(Signed) A. Branch-Manager.

The essential requirements for the form are: (1) a heading showing the name of the firm, department, unit, etc.; (2) the date(s) of the period under review; (3) space for entries for each individual absent (*a*) clock number, (*b*) name, initials and, for women, whether married or single (including widowed), (*c*) date of first and last day of absence and total number of days absent up to date, (*d*) reason for absence; (4) average number of persons employed during the period under review; (5) the form should be marked "confidential" and have a space for signature by the person responsible for filling it in.

In practice the form shown is perforated at the left-hand margin and is made up in book-form. The second sheet is a carbon copy of the original and is retained by the sender. Rest days are marked with an R and are not included in the cumulative totals of days absent from work, but, as they are important from the medical aspect, they are recorded by the clerk centrally in the penultimate right-hand column of the form, and added to the number of working days absent to give the "total days absent" in the final column.

On the form Mr. A. J. Brown had, at the beginning of the week, already been absent continuously for 37 working days and he has a further week's absence, making a total of 43 working days which will be inserted at the beginning of next week's return, i.e. in the column headed "days absent at start of week". This relates to an unbroken period of absence. Any return to work for even a day brings that period of sickness to an end. During this illness Brown has been ill on seven Sundays, so that his "total days absent" is now 50 days. Unless Mr. K. L. M. Jones returns to work on Monday he will start with four days absence at the beginning of the following week and he will also have lost one rest day by this time.

Women's entries are kept separately. (This is always desirable to avoid confusion but it is not essential where numbers are very small.) Mrs. P. Smith suffered concussion due to an accident away from work, and is therefore recorded as an accident off duty. All except Miss Robinson produced medical certificates, and the diagnosis on the certificate or the employee's statement is entered in the appropriate column. Since the names, etc., of all who were away on the last working day of this week will be again inserted in the first three columns of the following week, unless they return on the Monday, those who have been longest away will automatically appear at the head of the form, and thus a daily tally is kept of the numbers of days any individual is absent. This is of great use when a member is drawing sick benefit over a limited period and for sick clubs and visiting schemes which need a day-to-day knowledge of each worker.

Saturday, when worked, is counted as a whole day (and, of course, if not worked as R). Where the working population is varying the average number employed is obtained by adding the numbers who should be present (this includes the absentees) on the Monday to those who should be present on the Saturday (or Friday in a five-day week) and dividing by 2. Where the population is steady, the number who should be present at the beginning of the week is all that is necessary.

The Individual Records

Form A is the group record. For each worker it is desirable to have two records. One is the master or main record, the other the temporary or annual record.

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The master card consists of three parts: (1) means of identification of the employee; (2) unchanging (or infrequently changing) information essential for employment purposes; (3) other information for permanent record.

Under (1) are recorded the name and initials (and marital status for women), year of birth, and clock number. Sex is usually differentiated by the colour of the cards. Under (2) are recorded the employee's address (with space for two changes), date of entry to the firm, details of previous jobs and qualifications for the present one, grading, present occupation, and department. Sufficient space is left for promotions and other changes in status or work. Further spaces are required for recording whether the employee is a young person or a

disabled person. It is generally not practicable to record the rate of pay on this main card owing to frequent changes but a small firm may find it an advantage to do so. Under (3) are recorded, when known, important details of the individual's life, not necessarily confined to his industrial life; these will include any medical history and details of examinations made by a doctor for the firm. In this section, too, it is desirable to include the total annual amounts of sickness and accident absence which have occurred over the previous years.

A suggested design of this section is shown in Form B. Four main columns are required. The first records the calendar year, the second the numbers of days absent due to sickness and accident in that year, the third the number of absences in

FORM B

(Medical Section of Master Record)

SICKNESS AND ACCIDENT OCCURRING IN FORMER YEARS

Year	No. of Days Absent			No. of Absences	Accidents. Detail and whether AW or AF
	Working	Rest	Total		

the year, and the fourth gives brief details of the circumstances leading to, and nature of, each accident. Whether each was at work (AW) or not at work (AF) is also recorded. These summaries of sickness have proved of great value in large industrial concerns, e.g. the General Post Office, and are important in any concern when a medical officer undertakes to assess the health of an individual. The summaries from the current record are made at the beginning of each calendar year.

When employment ceases the main card passes to a wastage file, and, from this, labour turnover statistics can be compiled.

The Annual Record.—The annual record of the employee may, in the small firm, be filed with the main card and show only enough means of identification on it to link the two. It is, however, invariably simpler to compile statistics on sickness without reference to the main card. Thus the annual record, when not used in conjunction with the main record, requires: (1) means of identification (as on the main card); (2) year of birth; (3) grade; (4) department; (5) occupation. A suggested form is shown below (Form C).

Form C has been designed for easy alphabetical filing by name. As with the master card sex can be differentiated by colour, and the name should include initials for men and Christian name for women. The cards can be sorted by department

FORM C

Name of concern:	Year of birth:	Name:
Department:	Grade:	Marital status: M/S/W
Section:	Entry date: (to concern)	Clock No.:
Occupation:		

Periods of Absence

Date of Onset	Reason for Absence (Code for Sickness and Accident)	C or NC and AW or AF	Number of Days Absent			
			Working Days	Rest Days	Total Days	
					SA*	LA†
/ /5						
/ /5						
/ /5						
/ /5						
/ /5						
	(date) Annual Totals at	AW AF				

* SA: Short absence (three days or less).
† LA: Long absence (four days or more).

and section, occupation, year of birth, grade and marital status (women), and can be used, if wished, for monthly or quarterly periods by ruling off at the end of these times and adding sub-totals. Particulars of pay may be given under "grade". Sufficient space is allowed for at least one change under each heading "department", "section", "occupation", and "grade". If an individual changes his occupation within the concern more frequently than this in the year a new card should be issued. The actual day of the week on which any absence started can be added in the "date of onset" column if required. (Behrend has shown this to be of importance when setting a target to reduce absenteeism. It is, however, of less importance in sickness absence and is more readily obtained from Form A.)

On receiving information of new absences from Form A, or from the clock cards in smaller concerns, the card C is marked in columns 1 and 2 (date of onset and reason for absence) and then kept in hand until the sickness incident is complete, or until the 182nd day, when the remaining information is recorded. This includes, in column 3, recording whether the absence is certified or uncertified and, for accidents, whether AW or AF. The number of

working and rest days absent are inserted in columns 4 and 5 respectively, and the total days lost are divided into short absences of under four days (SA) or long absences of four days or more (LA). On completion the card is returned to the file. Each separate period of absence is thus entered and at the end of the year totals are made of their numbers (column 1), the numbers of accidents at work and not at work (AW and AF entries in column 3), the number of working days lost, the number of rest days " lost ", and the days lost due to short and long absences. This information is transferred to the main card, and the annual card, after statistical analysis, can be passed to a dead file.

Coding of Diagnoses

As medical certificates are frequently intended for perusal by lay persons they are couched in general or simple terms. Occasionally, sometimes deliberately, they are illegible, blank or obscure, but where a firm has the advice of a medical officer a high degree of accuracy in interpretation can be achieved ; since, however, the purpose here is to enable lay persons with no special knowledge to code the great majority of diagnoses, it is necessary to aim at a short list which is exhaustive for this particular purpose. Diagnoses which do not conform to the list given in the Appendix should, therefore, be placed in the miscellaneous group. This list includes diagnoses experienced by industrial and other concerns in London, the Home Counties, and the Midlands. There are, however, local variations in fashions in terms of diagnosis ; and in time any list becomes out of date. In addition, therefore, to the experience of the areas already mentioned, the present list was based on, and includes, the majority of diagnoses from the corresponding list given in Appendix B of the Industrial Health Research Board Report No. 85 (Med. Res. Coun., 1944), which was itself prepared from country-wide experience. Local additions, or sub-divisions, may be necessary, particularly where there are hazards not experienced by the majority ; for example, silicosis would be taken as a special sub-group of Group 5 (see Appendix) in those working groups exposed to such a hazard, while medical advice might well be sought if in any analysis more than about 10% of the diagnoses fall into the miscellaneous group.

The classification used is that of the International Statistical Classification of Diseases, Injuries, and Causes of Death (1948) with certain re-grouping of the components to provide essentially for minor morbidity in the industrial population. Any one using the full international code can readily re-group his data into the dozen categories suggested in the Appendix, and similarly anyone using this list could put the original coded data almost wholly into the international code.

The international code has 17 main sections to cover all modes of sickness and accident. Some of these are not applicable to an industrial population, e.g. diseases of infancy, and others, such as senility and neoplasms, are very seldom needed. For all normal purposes, therefore, it is suggested that 12 main groups will suffice. At the same time for each cause of absence the three-digit international code number is recorded as well, for it is from this that comparisons between concerns not using the present suggested system could be made, provided always that the other concerns have based their coding similarly on the international classification. The code figures placed against each diagnosis therefore are a single or two-digit group number followed by the three-digit international list number for that diagnosis ; for example, "pneumonia " falls in group 5 of the classification proposed here and its number in the international list is 493. Since some diagnoses must fall into the miscellaneous group, they cannot, from the following Appendix, be coded according to the international code ; it is important, therefore, that the diagnosis in words be recorded on the individual's documents, particularly when no code number has been allotted to it.

In the full international list accidents (Section XVII) are recorded both by the external circumstances giving rise to the injury, the E code, and by the nature of the injury, the N code ; the sub-group numbers are in each case 800–999. Only the N code is used in the present coding, and, since the numbers 800–999 thus refer only to the nature of the injury, they do not require the prefix N.

The international classification of disease is, in the main, anatomically determined, and this anatomical grouping has been retained and expanded in constructing the groups suggested for the present study. Thus certain sub-groups from Section I of the international classification (infective and parasitic diseases) have been removed from this group and placed under the anatomical site principally affected : for example, pulmonary tuberculosis, 002, has been allocated to the diseases of the lower respiratory system (present Group 5). Dysentery, similarly, has been added to the intestinal group (present Group 7). Most of the remaining diagnoses in this section have not been listed at all in the Appendix as their frequency in industry in this country is low. Similarly Section II of the international code (neoplasms) has not been classified

here, and certain conditions from Section III (allergic, endocrine, metabolic, and nutritional diseases) have been placed against their normal anatomical sites. For example, hay fever, 240, has been allotted to the upper respiratory system (Group 4) and asthma to the lower respiratory system (Group 5).

Sections IV and VII of the international classification (diseases of the blood and blood-forming organs and diseases of the circulatory system) have been combined to form Group I in the classification here proposed for use in industry. Sections V and VI become Groups 2 and 3 —with identical titles— while Section VIII (diseases of the respiratory system) has been here sub-divided into Groups 4 and 5 representing upper and lower respiratory disease respectively. Section IX (diseases of the digestive system) has similarly been sub-divided at the level of the duodenum. The upper part of the intestinal canal includes disorders of the mouth, (oesophagus), stomach, and duodenum in the present Group 6, and lower alimentary disease together with diseases of the abdominal organs in Group 7. Sections X and XI dealing with genito-urinary disease have been combined with pregnancy, childbirth, and the puerperium to form the present Group 8. Section XII (diseases of the skin and cellular tissue) becomes, with some additions relating to the lymphatic glands, the suggested Group 9, and Section XIII (diseases of the bones and organs of movement) forms the present Group 10. Accidents (Section XVII) are classified under Group 11 as AW or AF, it being unimportant from the point of view of medical classification whether these occurred at work or not, although, of course, the differentiation is of the utmost importance to industry. Group 12 is the miscellaneous group which covers all diagnoses not on the list, and blank and illegible certificates. In this group the three-digits 000 are coded after the group number 12.

Section XVI of the international classification contains " symptoms and ill-defined conditions ", and, since the medical certificate in industry may not infrequently record only such terms, e.g., vertigo, oedema, pleurodynia, they have been related to the appropriate anatomical group. Certain conditions which are quite commonly recorded on certificates are difficult to classify ; for example, phlebitis, unqualified, cannot strictly be coded, or, more often, having been coded, they are difficult to place appropriately in a group. Such terms as " black-out ", " chill ", " oedema ", and " headache " are not easily placed. Groups, however, have been found for them on the grounds that their relegation to the miscellaneous group would be a greater loss than would arise through a disagreement in grouping of a few individual diagnoses.

Any such classification is an exercise in eclectics, but the anatomical basis of disease is less often in doubt than is any alternative system, both from its nature, and, more important in the present study, from its coding by lay persons.

Small firms could shorten the grouping still further by combining Groups 4 and 5 into a single group of respiratory diseases and 6 and 7 into another of digestive diseases. Group 8 can be relegated to the miscellaneous group as it is always small and unnecessary when males only are employed. This would make a total of eight defined groups and one miscellaneous, and should suffice for valuable medical statistics to be gathered ; further reduction is inadvisable.

Statistical Analysis of the Data Recorded

The foregoing methods of recording have supplied the following data : (1) individual information on each person who has been absent, namely the number of days lost by short and long absences, the diagnosis of the cause of sickness and its diagnostic group and, in the case of an accident, whether it took place at or not at work ; (2) the numbers of men and women employed in the group under study, with sub-divisions, if required, by married and single women, certain age groups, etc.; (3) the number of days worked by the group ; (4) the total number of days lost by the group through sickness and accident ; (5) the number of persons who have been absent in relation to each diagnosed cause of sickness.

When using such data certain broad principles may be postulated as a guide. As a general rule he who initiates a scheme of recording should supervise the analysis, and the rates used should be easy to calculate and be easily appreciated by those who take action on them. It is, for instance, generally easier to interpret figures as percentages, but care must be taken that the numbers in the group are not so small as to make the percentage figure fallacious ; for example, one absent in a group of five would give a 20% absence rate but this figure is obviously not of the same significance as, say, 12 persons away in a group of 60. In another sense percentages can be misleading when, for example, the percentage of all absences falling in each diagnostic group is calculated. Since each kind of sickness is being expressed as a proportion of the total it follows that the total must be 100%. An epidemic of influenza might raise the percentage in Groups 4 and 5 from, say, 5% to 30%, and must then corres-

pondingly lower the percentage in some other groups, even though the absolute numbers of persons absent from these other causes is not lower.

It is clear that very small groups of under 20 or 30 persons should be compared only with caution or over a long period of time; preferably groups of 50 or more should be considered, and it may sometimes be necessary to group departments in order to achieve this. Finally as a general rule absences of men and women should not be considered together except to obtain an overall figure for a concern which may be desirable economically. These are merely commonsense points but they may serve to emphasize the need of care in the interpretation of results.

The essence of the statistical analysis is to find the numbers absent, and the time lost in relation to the numbers in, and the total time worked by, a particular department, section, etc. A concern, for instance, may wish to compare the average amount of time lost by short term sickness and accident as against long term sickness and accident. At the end of some specified period (usually month, quarter, or year) the number of days lost by short term absence and long term absence are separately aggregated. The cards are then sorted into the two sexes and four calculations are now required. The formula for the first may be expressed as :

$$\frac{\text{Total no. of days lost by men due to short absence in some specified period of time}}{\text{Average no. of men employed in the specified period of time}}$$

The remaining three calculations are made for long absence occurring amongst men, and long and short absence occurring amongst women in exactly the same way.

It may be desirable similarly to break down the figures arising from the example given below into the number of days lost by short and long term absence. If one of the groups shows more days lost than the others a more detailed investigation may be necessary into the types of sickness and accident suffered. (It should be remembered that in a small group one long-term illness may throw the figures out and if this occurs it should be noted.)

The following four calculations are thought to be the most useful for industrial management. First the average number of days lost per person through sickness and accident during some specified period—month, quarter, or year. This may be the total number of days lost or the number of working days lost. It may be sub-divided into younger and older men and younger and older women, into departments, etc. A concern, for example, may wish to compare the average amount of working time lost by sickness and accident in younger as against older employees. Some point of age may be chosen (say, 45 years) and the cards first sorted by sex and then by age. The first of the four calculations becomes :

$$\frac{\text{No. of working (or total) days lost by men under 45 years of age in the specified period}}{\text{Average no. of men under 45 years employed in the specified period}}$$

Following this the average number of working (or total) days lost by older men and older and younger women are now calculated.

Second, the loss of working time due to sickness and accident may be expressed as a proportion of the total time worked. The formula is :

$$\frac{\text{Total no. of working days lost}}{\text{Total no. of possible working days}} \times 100$$

The total number of possible working days is readily calculated by multiplying the number of persons in the group under study by the number of working days in the specified period. The total number of days lost is, of course, the aggregate of the individual losses (most easily obtained from Form A).

Third the percentage is calculated of persons who have been absent during a specified period. The formula is :

$$\frac{\text{Total no. of workers absent one or more times}}{\text{Average no. of workers employed}} \times 100$$

Fourth the average number of absences per worker during a specified period is required. The formula is :

$$\frac{\text{Total no. of absences}}{\text{Average no. of workers employed}}$$

It should be noted that in concerns where there are a number of grades comparisons between them may need to be made with considerable care if their conditions of employment vary. For instance, persons who have a month's annual holiday are at risk for 14 days less than those who have only a fortnight, those who work on Saturdays (counted as a whole day) are at risk for up to 52 extra days in the year. Strictly, of course, all these persons are at risk for the same period in regard to illness, but they are not necessarily recorded as being ill at week-ends or during their holidays.

In considering annual returns there is an advantage in studying the year from April 1 to March 31 rather than the calendar year. This is because epidemics occur about the turn of the calendar year, particularly of influenza and upper respiratory disease, the greatest causes of lost time in industry at this period. If an epidemic of influenza occurs late in the season, say January and February, 1952, and early in the following season, say November, December, 1952, then the incidence of sickness will

be much greater when calculated on a calendar year basis in 1952 than in either 1951 or 1953.

The Presentation of Results

Complete information is now available and it remains only to suggest suitable means of presentation of the results.

Classification of Causes of Sickness and Accident.—Form D is designed to show, after the name of the department concerned and the dates of the period under review, the absolute number of cases starting in the period under review, the number of cases continuing into that period, and the number of days lost, for men and women separately and for each diagnostic group. The last column shows the percentage of the total loss (100%) due to each diagnostic group, calculated as follows :

$$\frac{\text{No. of days lost by each sex in diagnostic Group X}}{\text{Total no. of days lost by each sex in all diagnostic groups}} \times 100$$

Quarterly and annual returns are made by aggregating monthly totals and re-calculating the percentages.

Analysis of Sickness and Accident by Sex and Department.—A suitable form for setting out the analysis of absence by sex and department is given in Form E.

Column 1 shows the sex and column 2 each department by name or code. The average number employed (column 3) is obtained by averaging the weekly aggregates, and the total number of working days (column 4) is the number of persons in the department multiplied by the number of working days in the period under review. Column 5 shows the actual number of persons who were absent one or more times, and column 6 the total number of days they lost. Column 7 gives the average number of days lost per person employed, i.e. column 6 divided by column 3.

FORM D

CLASSIFICATION OF CAUSES OF SICKNESS AND ACCIDENTS

Dept..... .....195....

Diagnostic Group	No. of New Cases		No. of Continuing Cases		Days Lost		Percentages of Total Loss	
	Men	Women	Men	Women	Men	Women	Men	Women
1. Circulatory ..								
2. Mental ..								
3. Nervous ..								
4. Upper respiratory ..								
5. Lower respiratory ..								
6. Mouth and stomach ..								
7. Abdominal and intestinal ..								
8. Genito-urinary								
9. Skin								
10. Muscle, bone and joint ..								
11. Accident : AW Accident : AF								
12. Unclassified..								
Total ..							100	100

New cases are those beginning between midnight on the last day of the previous period and midnight on the last day of the current period. Continuing cases are those beginning before the current period.

Column 8 gives the percentage loss of time and is obtained by dividing column 6 (days lost) by column 4 (no. of working days) and multiplying by 100. Finally column 9 shows the percentage of persons absent, i.e., column 5 divided by column 3 then multiplied by 100.

FORM E

Name of Concern..... Period.....

ANALYSIS OF SICKNESS AND ACCIDENT ABSENCE BY SEX AND DEPARTMENT

Sex (1)	Department (2)	Average No. Employed (3)	Total No. of Working Days (4)	No. of Persons Absent (5)	No. of Days Lost (6)	Average No. of Days Lost per Person Employed (7)	Percentage Loss of Time (8)*	Percentage of Persons Absent (9)
Male ..								
Total ..								
Female..								
Total ..								
Total M. + F.								

* State whether a five- or six-day week.

This form gives on a single page a useful summary of the health of a concern over a specified period, and should reveal where further detailed analysis, e.g. by age and diagnostic cause, is called for and where preventive action against ill health or accident needs to be taken.

I am greatly indebted to Prof. Bradford Hill, C.B.E., for advice and constant help throughout, and to the members of the Medical Research Council's Occupational Health Committee individually for advice and helpful criticism. Thanks are also due to Mr. F. J. Lloyd, Dr. L. G. Norman, and Dr. P. A. B. Raffle, of the London Transport Executive, on whose work much of this study depends, and to the concerns listed in I.H.R.B. Report No. 85 for putting their valuable experiences at my disposal.

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APPENDIX

Group No.	Name of Group	International Classification
1	Diseases of the circulatory system, blood, and blood-forming organs	IV 293 VII 410 420 422 433–4 443–4 453 460–1 463 466 XVI 782
2	Mental, psychoneurotic, and personality disorders	V 310 315–6 318 XVI 790–1
3	Diseases of the nervous system and sense organs	I 088 VI 353–4 360 363 366 370–3 381 391 394 XVI 780
4	Upper respiratory disease	I 051 070 III 240 VIII 470–4 481–2 511–2 514–5
5	Lower respiratory disease	I 002 III 241 VIII 480 490–3 501 518–9 522 527 XVI 783
6	Diseases of mouth, stomach, and duodenum (upper alimentary tract)	I 048–9 IX 530–4 536 538 540–1 543–4 XVI 784
7	Diseases of abdominal organs and intestinal tract	I 092 IX 551 560 571–2 574 584–5 XVI 785
8	Diseases of genito-urinary system, disorders of pregnancy, child-birth, and puerperium	X 593 600 605 613–4 630 634 637 XI 649 XVI 789
9	Diseases of skin, cellular tissue, and lymphatic glands	I 052 131 III 243 XII 690–6 700–3 705–6 708 712 714–5 XVI 786 788
10	Diseases of bones, joints, and organs of movement	III 288 XIII 722–3 725–7 734 738 740–2 746–7 787
11	AW. Accidents AF. (In the list of diagnoses the N prefix is not given and is not coded)	NXVII N803 N807 N817 N824–6 N829 N839 N842 N845 N850–2 N869 N879 N883–4 N985 N914–5 N917–8 N928–30 N941 N944 N949
12	Miscellaneous	All sections II, XIV, and XV and parts of sections I, III–XIII, XVI, and XVII not included above

CODING OF COMMON ILLNESSES

A

Abscess	.. ..	9	692
Abscess of ear	.. ..	3	391
Adenitis, acute	..	9	694
Alveolar abscess	..	6	531
Anaemia	.. ..	1	293
Anal fissure, fistula	..	7	574
Angina pectoris	..	1	420
Anxiety state	.. ..	2	310
Appendicitis	.. ..	7	551
Arrythmia, cardiac	..	1	433
Arthritis	.. ..	10	725
Asthenia	.. ..	2	790
Asthma	.. ..	5	241
Athlete's foot	.. ..	9	131

B

Backache	.. ..	10	787
Bell's palsy	.. ..	3	360
Bilious attack	.. ..	6	544
Blackout	.. ..	1	782
Bladder, diseases of	..	8	605
Blepharitis	.. ..	3	371
Boil or carbuncle	..	9	690
Bronchial asthma	..	5	241
Bronchial catarrh	..	5	501
Bronchitis	.. ..	5	501
Bronchopneumonia	..	5	491
Bruise, see Contusion			
Buccal sepsis	.. ..	6	538
Bunion	.. ..	10	740
Burn, face, head	..	11	941
hand, wrist	..	11	944
other	.. ..	11	949
Bursitis	.. .. ..	10	741

C

Carbuncle or boil	..	9	690
Cardiac debility	..	2	315
Caries, dental	.. ..	6	530
Catarrhal cold, fever	..	4	470
Catarrhal jaundice	..	7	092
Cellulitis	.. ..	9	692
Cephalgia	.. ..	2	791
Cervicitis	.. ..	8	630
Cheiropompholyx	..	9	701
Chilblains	.. ..	1	453
Chill	.. .. ..	9	788
Cholangitis	.. ..	7	585
Cholecystitis	.. ..	7	585
Cold	.. .. ..	4	470
Colic	.. .. ..	7	785
Colitis	.. ..	7	571
Concussion	.. ..	11	852
Congestion of lungs	..	5	522
Conjunctivitis	..	3	370
Contusion, foot, toes	..	11	928
scalp	..	11	851
other	..	11	929
Corneal ulcer	.. ..	3	381
Coronary disease	..	1	420
Coryza	.. ..	4	470
Cough	.. .. ..	5	783
Cut, see Laceration			
Cyst, sebaceous	..	9	714
Cystitis	.. .. ..	8	605

D

Debility	.. ..	2	790
Dental abscess	..	6	531
Depression	.. ..	2	790
Dermatitis	.. ..	9	703
Dermatophytosis	..	9	131
Diarrhoea	.. ..	7	785
Dislocation	.. ..	11	839
Disorder of menstruation	.. .. ..	8	634
Diverticulitis	.. ..	7	572
Duodenal ulcer	..	6	541
Duodenitis	.. ..	6	543
Dysentery	.. ..	6	048
Dysmenorrhoea	..	8	634
Dyspepsia	.. ..	6	544
Dyspepsia, nervous	..	2	316

E

Earache	.. ..	3	394
Eczema	.. ..	9	701
Emesis	.. .. ..	6	784
Emphysema	.. ..	5	527
Empyema	.. ..	5	518
Endocervicitis	..	8	630
Enlarged glands	..	1	782
Enteritis	.. ..	7	571
Epidermophytosis	..	9	131
Epididymitis	.. ..	8	614
Epilepsy	.. ..	3	353
Epistaxis	.. ..	5	783
Erysipelas	.. ..	9	052
Erythema	.. ..	9	795
Eye, diseases of	..	3	370

F

Facial paralysis	..	3	360
Fainting attack	..	1	782
Fatigue	.. ..	2	790
Fibrositis	.. ..	10	726
Flat feet	.. ..	10	746
Fluid of knee	.. ..	10	742
Food poisoning	..	6	049
Foreign body in eye	..	11	930
Fracture ankle	..	11	824
foot	..	11	825
hand	..	11	817
ribs	..	11	807
skull	..	11	803
toes	..	11	826
other	..	11	829
Furunculosis	.. ..	9	690

G

Gall stones	.. ..	7	584
Gastric condition	..	6	540
Gastric influenza	..	4	482
Gastric ulcer	.. ..	6	540
Gastritis	.. ..	6	543
Gastro-enteritis	..	7	571
Giddiness	.. ..	3	780
Gingivitis	.. ..	6	532
Glands, swollen	..	1	782
Glossitis	.. ..	6	538
Gout	.. .. ..	10	288
Graze, see Injury, superficial			
Grippe	.. .. ..	4	481
Gums, inflammation of		6	532

H

Haematemesis	..	6	784
Haematuria	.. ..	8	789
Haemoptysis	.. ..	5	783
Haemorrhoids	..	1	461
Hallux valgus	.. ..	10	747
Hay fever	.. ..	4	240
Headache	.. ..	2	791
Heart disease	.. ..	1	434
Heartburn	.. ..	6	784
Hepatitis, infectious	..	7	092
Hernia, inguinal or femoral	.. ..	7	560
Herpes zoster	.. ..	3	088
High blood pressure	..	1	443
Hordoleum	.. ..	3	372
Hydrocoele	.. ..	8	613
Hyperpiesis	.. ..	1	444
Hypertension	.. ..	1	444

I

Impacted tooth	..	6	533
Impetigo	.. ..	9	695
Indigestion	.. ..	6	544
Industrial dermatitis	..	9	702
Infectious hepatitis	..	7	092
Inflamed eye	.. ..	3	370
Influenza	.. ..	4	481
Influenzal pneumonia		5	480
Injury, internal	..	11	869
Iritis	.. .. ..	3	373
Injury, superficial :			
fingers	..	11	915
foot and toes	..	11	917
hands	.. ..	11	914
other	..	11	918
Ingrowing toenail	..	9	712
Internal derangement of knee	.. ..	10	734
Intestinal colic	..	7	785
Iritis	.. .. ..	3	373

J

Jaundice	.. ..	7	785

K

Kidney infection	..	8	600
Kidney trouble	..	8	593
Knee cartilage	.. ..	10	734

L

Laceration, face, neck, trunk	..	11	879
finger	..	11	884
hand	..	11	883
lower limb		11	895
scalp	..	11	850
Laryngitis	.. ..	4	474
Leucorrhea	.. ..	8	637
Lobar pneumonia	..	5	490
Lumbago	.. ..	10	726
Lymphangitis	.. ..	9	693

M

Menorrhagia	.. ..	8	634
Menstrual disorder	..	8	634
Migraine	.. ..	3	354
Mitral stenosis	..	1	410
Mouth, diseases of	..	6	538
Muscular rheumatism		10	726
Myalgia	.. ..	10	726
Myocarditis	.. ..	1	422
Myositis	.. ..	10	726

N

Nasal catarrh	.. ..	4	512
Nasal polypus	.. ..	4	515
Nasopharyngitis, acute or N.O.S.	.. ..	4	470
Nephritis	.. ..	8	593
Nerves	.. .. ..	2	790
Nervous breakdown	..	2	318
Nervous debility or exhaustion	.. ..	2	318
Nervous dyspepsia	..	2	316
Nettlerash	.. ..	9	243
Neurasthenia	.. ..	2	318
Neuritis	.. ..	3	366
Neurosis	.. ..	2	318

O

Oedema	.. ..	1	782
Onychia	.. ..	9	691
Orchitis	.. ..	8	614
Osteo-arthritis	.. ..	10	723
Otitis media or otorrhoea	.. ..	3	391

P

Paronychia	.. ..	9	691
Peptic ulcer	.. ..	6	540
Peripheral neuritis	..	3	366
Pharyngitis	.. ..	4	472
Pes planus	.. ..	10	746
Phlebitis	.. ..	1	463
Phthisis	.. ..	5	002
Pleural effusion	..	5	519
Pleurisy	.. ..	5	519
Pleurodynia	.. ..	5	783
Pneumonia	.. ..	5	493
Pneumonitis	.. ..	5	492
Pregnancy, conditions in	.. .. ..	8	649
Pruritus	.. ..	9	708
Psoriasis	.. ..	9	706
Psychoneurosis	..	2	318
Pulmonary tuberculosis		5	002
Pyelitis	.. ..	8	600
Pyelonephritis	..	8	600
Pyorrhea	.. ..	6	532
Pyrexial catarrh	..	4	470

Q

Quinsy	.. ..	4	511

R

Rash	.. .. ..	9	788
Raynaud's disease	..	1	453
Renal colic	.. ..	9	786
Rheumatism	.. ..	10	727
Rheumatoid, any condition	.. ..	10	722
Rhinitis, acute	..	4	470
Rupture	.. ..	7	560

S

Sciatica	.. ..	3	363
Seborrhea	.. ..	9	700
Septic finger, toe	..	9	691
Septic throat	.. ..	4	051
Septum, nasal, deflected		4	514
Shingles	.. ..	3	088
Shock	.. .. ..	1	782
Sickness and diarrhoea		7	571
Sinusitis, acute	..	4	471
Sore throat	.. ..	4	472
Spondylitis	.. ..	10	723
Sprain or strain :			
ankle, foot	.. ..	11	845
wrist, hand	.. ..	11	842
Stiff neck	.. ..	10	726
Stomach ulcer	.. ..	6	540
Stomatitis	.. ..	6	536
Strained heart	..	1	434
Stye	.. .. ..	3	372
Swollen glands	..	1	782
Syncope	.. ..	1	782
Synovial effusion	..	10	738
Synovitis	.. ..	10	741

T

Tachycardia	.. ..	1	782
Tenosynovitis	.. ..	10	741
Tinea pedis	.. ..	9	131
Toothache	.. ..	6	534
Tonsillitis, acute	..	4	473
Thrombosis	.. ..	1	466
Tracheitis, acute	..	4	474
Tuberculosis, pulmonary	.. .. ..	5	002

U

Ulcer of skin	.. ..	9	715
Ulcer of duodenum	..	6	541
Ulcer of stomach	..	6	540
Urinary infection	..	8	600
Urticaria	.. ..	9	243

V

Varicose ulcer of veins		1	460
Verruca	.. ..	9	696
Vertigo	.. .. ..	3	780
Vincent's angina (or infection)	.. ..	4	070
Vomiting	.. ..	6	784

W

Warts	.. .. ..	9	696
Wen	.. .. ..	9	714
Whitlow	.. ..	9	691

Code all other diseases as Group 12 (Miscellaneous)

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SOME EFFECTS OF PAID SICK LEAVE ON SICKNESS ABSENCE

BY

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In recent years the tendency to give greater economic protection to industrial workers has led to the introduction of many sickness benefit schemes. This article considers the effects on sickness absence of two schemes for the payment of wages during illness and injury.

Company Z was a large organization controlling many factories in different parts of the country. In September, 1948, workers with at least six months' service were granted sick leave with full pay for a maximum of 13 weeks in any 12 months, and those with at least five years' service were granted an additional period of 13 weeks on half pay. "Full pay" was defined as ordinary time rate minus the amount of National Insurance benefit to which the worker was entitled. Medical certificates were required after three days of sickness or injury, and subsequently at weekly intervals. Payment was not made for the first three days' absence unless the incapacity lasted for at least five working days.

Company V, another large industrial organization with many factories throughout the country, had operated since 1928 an establishment scheme giving "to workers selected on grounds of individual merit a status and a form of security approximating to that of staff". An established worker was guaranteed a weekly wage at least equivalent to his ordinary time rate, and if sick or injured, received this amount (less National Insurance benefits) for a maximum of 26 weeks in any calendar year. He was also entitled to four weeks' notice of the termination of his employment, and, in addition, could be granted leave of absence on full pay at the discretion of the management. All workers who had completed three years' service and were at least 24 years of age were eligible for establishment but not more than half of them could be in the established grade at any one time. Selection depended upon the assessment of certain personal qualities, and each eligible worker was rated by his local management every six months. Various methods were used in different parts of the organization, but the qualities assessed usually included willingness, industry, initiative, reliability, example to others, ability, and adaptability. Very little weighting was given to length of service, but the worker's timekeeping record was considered. It was laid down that absence due to certified sickness should not affect a worker's chances of being established. An established worker was paid for all absence of less than three days' duration provided that a satisfactory explanation was given; medical certificates were required on the third day of absence and subsequently at weekly intervals. Complementary to the establishment scheme, all non-established men who had completed three years' service qualified automatically for sick pay at the rate of 12s. 6d. per week for a maximum of 13 weeks in any year.

Absence in Company Z

Company Z employed over 40,000 workers, almost 90% of whom were men. The percentages of working days lost due to certified sickness (including injury) during the 12 months before and after the introduction of sick leave with pay are shown in Fig. 1. Absence curves are also included for all male employees of Company V, the 20,000 established workers being shown separately from the 41,000 non-established workers. The figures on which the graphs are based are given in Table A of the Appendix.

Before Company Z introduced sick leave with pay absence was closely similar to that among non-established workers in Company V. As soon as the scheme was introduced, there was an immediate and spectacular increase in absence, and during the following year it was at a level comparable with that of established workers in Company V. Both curves for Company V followed the usual seasonal

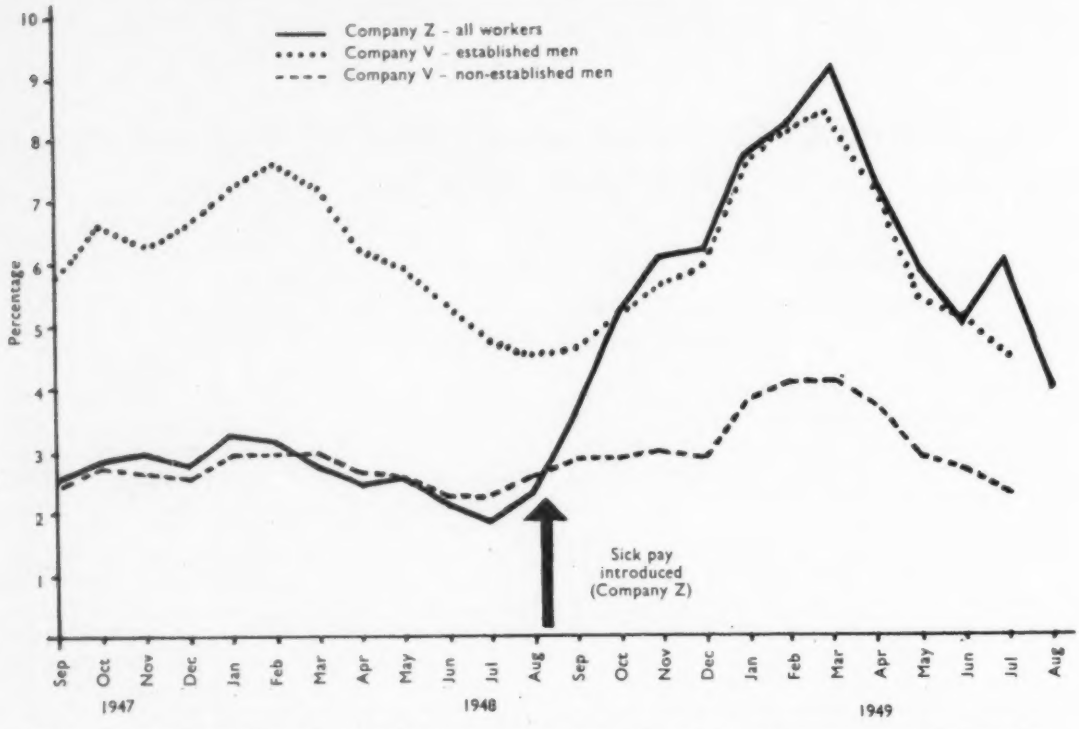
Company Z - all workers
Company V - established men
Company V - non-established men
Percentage
Sick pay introduced (Company Z)
Sep Oct Nov Dec Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec Jan Feb Mar Apr May Jun Jul Aug
1947
1948
1949

FIG. 1.—Sickness absence before and after the introduction of paid sick leave (no. of shifts lost as percentage of no. of shifts possible).

trends, and during the winter of 1948–49 absence was about 1% higher than in the previous year. As there was no radical change in the composition of Company Z's labour force, it seems reasonable to expect that it, too, would have shown a similar small increase if the sick pay scheme had not been introduced. Absence was, however, more than doubled compared with the previous year, and it seems certain that most of this increase was attributable to sick leave with pay. Some reasons for this are discussed later in this paper.

In Company Z it was also possible to demonstrate the influence which financial incentives can exert on sickness absence. In each of two factories (X and Y) there was a department of about 150 men working under closely similar conditions. The factories were in different parts of the country, but the two departments had started work in 1946 and they were practically identical as regards lay-out, equipment, and production methods. The men in Department Y of Factory Y were, however, older and physically less fit than those in Department X of Factory X. Both departments were paid on group output bonus systems which were similar in principle, but since 1947 the men in Department Y had been dissatisfied with their earnings and had claimed that the bonus standards were too tight. In consequence there was an atmosphere of mutual distrust between management and workers, and relations were not so good as in Department X. It was not, therefore, surprising that during 1947–48 the men in Department Y usually had more absence than those in Department X, and, in particular, they were more prone to take occasional days off from work.*

Both departments were scheduled to cease production at the end of 1948, and the men were to be offered jobs in other parts of the factories. The circumstances under which the departments closed were, however, quite different. The men in Department X were told of the approaching end of production in November, and during the following weeks some of the regular workers were transferred to a new department. They were replaced by trainees who could not be expected to have much enthusiasm for a job which was drawing to its close. In contrast to this the men in Department Y

* During the 12 months ending September 4, 1948, absence due to all causes averaged 7·2% in Department Y as compared with 5·8% in Department X.

were warned in August that production would cease at the end of the year, and they were given the opportunity to take over the uncompleted contracts of other factories. An assurance was given by the management that the bonus scheme would not be altered however high the earnings rose. The men in both departments knew that when production stopped they would be transferred to other work with no chance of earning bonuses for several weeks. Accordingly the men in Department Y decided to go all out while the going was good, and the output bonus rose to £3 15s. per man per week, which was about twice that being received by the men in Department X. Fig. 2 shows the weekly absence in these two departments during the latter half of 1948; comparable data are also included for all men employed in Factory Y. Reasons for absence having been classified less accurately in Factory Y than in Factory X, it has been necessary to compare figures of absence due to all causes*. The data on which Fig. 2 is based are given in Table B of the Appendix.

throughout the first quarter of 1949 an average of one man in five was away from work. But the figures for Department Y did not show any corresponding increase, and absence at the end of the year was the same as it had been in September when sick pay was introduced. It would seem that for these men any temptation to take advantage of sick leave with pay was neutralized by the lure of high earnings. During this period minor ailments were ignored and some men came to work when they appeared unfit to do so. Typical comments from workers who were interviewed were :—

> "We're getting a good bonus now, but I doubt whether the men can keep this pace going. It's too much for some of them, but they want the money."
>
> "Some of the men aren't fit to be at work, but they know what's in store for them, and they're taking their chance whilst they can."

The financial incentive and the pace of work were abnormally high, and neither could have been maintained for long. In fact there were signs of a sharp increase in absence during the last week of December when work was fading out. The men

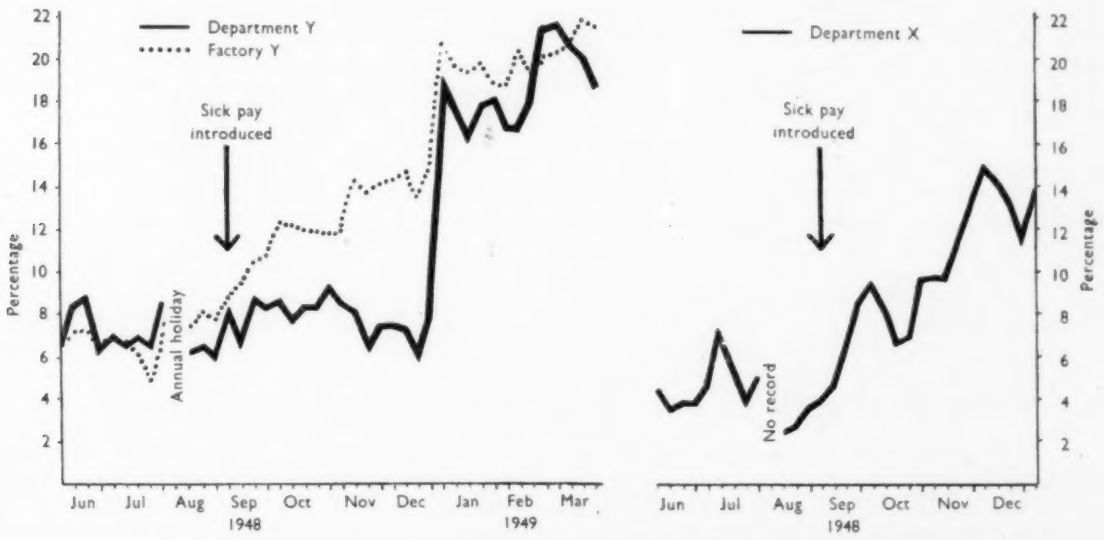

FIG. 2.—Weekly absence due to all causes (no. of shifts lost as percentage of no. of shifts possible).

In Department X the introduction of sick pay was immediately followed by a rapid increase in absence. From November onwards, when it was known that the department was to close, absence due to all causes averaged about 13%. In Factory Y there was also an immediate and spectacular rise in the absence rate. By the end of the year absence was twice what it had been in December, 1947, and were transferred to other departments at the beginning of January, and their absence records were obtained for the following three months. It will be observed that when the spur of exceptionally high earnings was removed the rate of absence immediately climbed to the high level which existed in the rest of Factory Y. In these circumstances, it seems likely that the need to earn as much as possible before the department closed had kept

* A more detailed explanation is given on page 279.

these men at work when they might otherwise have stayed away. The fact that they did not succumb to the temptation of sick leave with pay suggests the extent to which minor ailments can temporarily be resisted when the financial incentive is sufficiently strong.

Absence in Company V

Further reference to Fig. 1 shows that between 1947 and 1949 sickness absence among established men in Company V was roughly double that among non-established. Sickness has always been higher among established workers, and even during the pre-war years when there was considerable unemployment and hence less security, it was often 50% greater. Shortly after the war the gap between the figures widened, absence having increased more among the established men than among the non-established. In comparing these figures it is important to take into account the effects of age on sickness absence, as the established workers were on the average about 10 years older than the non-established.

In one of Company V's factories, Factory C, where about 1,000 established and 2,500 non-established men were employed, an analysis was made of sickness absence during the period from January to June, 1948. The figures for each age group are shown in Table 1.

ment of wages during sickness enabled workers to stay at home until fully recuperated so that some quite justifiable extension of sickness absence could be expected*. It also seems reasonable to assume that in some cases doctors prescribed treatment or convalescence which were only feasible for workers whose earnings were protected. It is probable, too, that some workers took sick leave when they might otherwise have remained at work. These consequences of sick leave with pay would result in a higher absence rate, but the ensuing benefits to health should help to reduce the risk of sickness in later years.

Table 1 shows that the greatest difference in sickness absence between the established and non-established workers was in the 25 to 34 age group, and the least difference was between workers above the age of 55. If the non-established men were unable to stay away from work when sickness would have justified them doing so, or if they returned to work before they were fit, they might be expected to incur more frequent or more prolonged absence later in their working lives. Although the percentage differences in sickness absences do not show a consistent trend (see Table 1), the small difference between workers over the age of 55 may be partly due to the fact that the payment of wages during previous sickness had to some extent

TABLE 1
RELATION BETWEEN AGE AND SICKNESS ABSENCE IN FACTORY C JANUARY TO JUNE, 1948

Age Group	Established Men				Non-established Men				Percentage Difference
	Number of Men	Shifts Possible	Shifts Lost		Number of Men	Shifts Possible	Shifts Lost		
			Number	%			Number	%	
Under 25	—	—	—	—	235	29,845	543	1·82	—
25 to 34	127	16,129	560	3·47	664	84,328	1,527	1·81	1·66
35 to 44	355	45,085	1,417	3·14	652	82,804	1,889	2·28	0·86
45 to 54	392	49,787	2,072	4·16	552	70,104	2,048	2·92	1·24
55 and over	209	26,513	1,440	5·43	391	49,657	2,632	5·30	0·13

In both groups sickness absence tended to increase with age*. Hence the established workers in Factory C could be expected, because of their higher average age, to have a higher absence rate than the non-established workers. Although absence figures according to age were not available for Company V as a whole, it seemed reasonable to assume that, as in Factory C, the higher average age of the established men was one reason for their greater absence.

But, in each case when men of the same age were compared, the established men had more absence than the non-established (see Table 1). The pay-

protected the health of the established workers.

It does however seem certain that there was some deliberate abuse of sick leave with pay. Some evidence in this connexion was provided by the results of an attitude survey in Factory C, where, amongst other questions, each worker was asked, " Are you in the established grade ? " and, " What do you think of it ? " About 15% of both the established and the non-established men said that the scheme was abused by some workers. The

* A similar trend was shown by absence records obtained from another large organization.

* During the enquiries in Factory C, 22 established and 34 non-established men resumed work after periods of certified sickness ; 18 of the former and 20 of the latter returned on the first day of the week, and not one person restarted after the third day of the week. Although this evidence is scanty, it would seem that some doctors tend to sign off patients so that they can resume work at the beginning of a week.

former tended to point out that " only a few workers take advantage of the scheme ", whereas the latter more often referred in general terms to its abuse. In one large department where 35 supervisors were interviewed, 31 of them said that there was some abuse of the scheme. The absence rates in Factory C at this period compared favourably with absence in Company V as a whole. Analysis of records maintained by Factory C indicated that absence among established workers tended to be both more frequent and more prolonged than amongst non-established workers. Referring again to Table 1 it will be observed that established men below the age of 35 had rather more absence than those between the ages of 35 and 44. A higher proportion of the younger age group would have been recently established and there are grounds for surmising that newly promoted workers were especially prone to take advantage of the scheme. In discussing this aspect of establishment, one workers' representative said, " We've got some fellows who never had a day's illness in their lives until they were promoted ". Thus, some abuse of the scheme existed even though the selection of workers was based on qualities such as " reliability " and " example to others ".

Discussion

In both these companies the granting of sick leave with pay to workers whose earnings had not previously been protected was followed by an increase in sickness absence. Company Z introduced sick pay only two months after the National Health Service started, and the combination of these two schemes radically altered the financial position of workers during sickness, especially of those with families to support. Workers who had been unable to afford operations or long courses of treatment were likely to take an early opportunity of having them under the National Health Service, particularly as Company Z guaranteed to pay their full time-rate while they were away from work. It seems probable that the rapid increase in sickness absence experienced by Company Z was partly due to the fact that the sick pay scheme was introduced so soon after the National Health Service.

Absence in Department Y demonstrated that the temptation to take advantage of sick leave with pay could be resisted when the financial incentive was exceptionally strong. It also seems reasonable to assume that temptation to malinger would be particularly great when the worker was financially better off at home than at work. In this respect it should be noted that during sickness a worker paid no National Insurance contribution and no income tax on his National Insurance sickness benefit. The compulsory deductions from wages might, therefore, be very much less than those usually paid. In addition, some workers received sickness benefits from friendly societies to which they voluntarily contributed.

The greater incentive to obtain medical certificates led in some factories to more accurate classification of the reasons for absence. In Factory Y total absence had averaged 7·2% during the year before sick pay was introduced, and of this 3·2% had been classified as certified sickness and 3·3% as absence without leave. In the following year absence without leave dropped to 2·0% and sickness absence rose to 12·3%, representing 81% of the total absence as compared with only 44% formerly. There had previously been no systematic enforcement of the rule requiring medical certificates to be produced after three days of absence, and there can be no doubt that some cases of genuine sickness had been recorded as absence without leave. Hence, a comparison of the recorded sickness rates for these two years would exaggerate the increase attributable to sick leave with pay. By comparison, absence had been more efficiently classified in Factory X, and during these two years absence without leave showed no significant reduction.

In Company Z workers were not paid for the first three days' sickness unless their absence lasted for at least five working days. There was thus a strong temptation to prolong short illnesses. Established workers in Company V were paid for all absence and it seems probable that this encouragement to obtain early treatment may have prevented some longer or more serious illnesses and reduced the risk of spreading infection in the factory. Both schemes removed much of the financial worry and anxiety which many workers had previously associated with sickness absence. Typical comments by men interviewed in Factory C were :—

> " It gives a sense of security during illness and removes the nagging fear about your family."
>
> " It gives a man security—a month's notice, and his wages when sick. Some men used to start back at work before they were really better."

On the other hand, although most workers appreciated the reduced economic pressure to resume work, there were others for whom the inclination to work was also lessened. Some men stayed away from work unnecessarily, and others prolonged minor ailments for the purpose of obtaining sick pay. In Company Z several men who had obtained medical certificates from their doctors were found to be drawing sick pay

when working temporarily for other employers.

This article has been confined to the effects on sickness absence of two schemes for the payment of wages during illness. It seems probable that, among other important effects, these schemes may give rise to increased feelings of satisfaction and loyalty to the firm, but such considerations are not within the scope of this article.

Summary

In Company Z sickness absence rose from 2·6% in the year before the introduction of sick pay to 6·1% in the year after it was introduced (Table A). In one department, where bonus earnings were exceptionally high, the introduction of sick pay had no effect on sickness absence until earnings were substantially reduced (Table B).

In Company V sickness absence for established men was roughly double that for non-established men (Table A). Some of this increase was attributable to the higher average age of the established men, but when age was made constant, the difference in sickness absence was still appreciable (Table 1). There was evidence that a few established workers abused the scheme, particularly those who had only recently begun to benefit from it.

Payment of wages during illness reduced the workers' fear of sickness absence, and may have helped to prevent further sickness by providing the opportunity to recuperate fully before resuming work.

APPENDIX

TABLE A

PERCENTAGE OF SHIFTS LOST DUE TO CERTIFIED SICKNESS AND INJURY

Month		Company Z	Company V		Month		Company Z	Company V	
			Established	Non-established				Established	Non-established
1947	September	2·5	5·8	2·4	1948	September	3·5	4·6	2·8
	October	2·8	6·6	2·7		October	5·1	5·1	2·8
	November	2·9	6·3	2·6		November	6·0	5·6	2·9
	December	2·7	6·6	2·5		December	6·1	5·9	2·8
	January	3·2	7·2	2·9		January	7·6	7·5	3·7
1948	February	3·1	7·6	2·9	1949	February	8·1	8·1	4·0
	March	2·7	7·2	2·9		March	9·0	8·3	4·0
	April	2·4	6·2	2·6		April	7·1	7·1	3·6
	May	2·5	5·9	2·5		May	5·8	5·3	2·8
	June	2·1	5·3	2·2		June	4·9	5·0	2·6
	July	1·8	4·7	2·2		July	5·9	4·4	2·2
	August	2·2	4·5	2·5		August	3·9	No record	
Average		2·6	6·2	2·6	Average		6·1	6·1	3·1

TABLE B

WEEKLY ABSENCE DUE TO ALL CAUSES

Week Ending	Factory Y			Department Y			Department X		
	Possible Shifts	Shifts Lost		Possible Shifts	Shifts Lost		Possible Shifts	Shifts Lost	
		Number	%		Number	%		Number	%
5. 6.48	9,335	612	6·6	930	61	6·6	716	31	4·3
12. 6.48	9,320	675	7·2	925	78	8·4	735	25	3·4
19. 6.48	9,300	666	7·2	920	82	8·9	735	27	3·7
26. 6.48	9,270	601	6·5	905	58	6·4	735	27	3·7
3. 7.48	9,270	654	7·0	880	62	6·9	730	34	4·7
10. 7.48	9,260	622	6·7	880	57	6·5	686	49	7·1
17. 7.48	9,260	562	6·1	880	61	6·9	705	38	5·4
24. 7.48	9,250	457	4·9	880	56	6·4	649	24	3·7
31. 7.48	9,250	702	7·6	880	76	8·6	723	36	5·0
7. 8.48	Factory closed for holiday week						No record		
14. 8.48	9,255	694	7·5	880	54	6·1	704	17	2·4
21. 8.48	9,255	757	8·2	875	57	6·5	659	17	2·6
28. 8.48	9,225	719	7·8	870	51	5·9	650	22	3·4
4. 9.48	9,230	816	8·8	865	72	8·3	676	26	3·8
11. 9.48	9,225	877	9·5	865	57	6·6	696	31	4·5
18. 9.48	9,190	971	10·5	870	76	8·8	682	43	6·3
25. 9.48	9,165	990	10·8	875	72	8·2	682	57	8·4
2.10.48	9,125	1,116	12·3	940	81	8·6	726	68	9·4
9.10.48	9,105	1,106	12·2	915	69	7·6	738	59	8·0
16.10.48	9,080	1,085	12·0	915	75	8·2	740	48	6·5
23.10.48	9,055	1,080	11·9	915	75	8·2	743	51	6·9
30.10.48	9,040	1,066	11·8	915	86	9·4	602	57	9·5
6.11.48	9,140	1,088	12·0	915	79	8·6	585	56	9·6
13.11.48	9,150	1,311	14·3	910	75	8·2	565	54	9·6
20.11.48	9,090	1,252	13·8	910	57	6·3	584	66	11·3
27.11.48	9,085	1,286	14·2	910	68	7·5	575	75	13·0
4.12.48	9,090	1,307	14·4	905	68	7·5	565	83	14·7
11.12.48	9,090	1,337	14·7	900	67	7·4	559	78	13·9
18.12.48	9,090	1,240	13·6	900	53	5·9	565	73	12·9
25.12.48	7,260	1,079	15·1	720	57	7·9	444	50	11·2
1. 1.49	7,244	1,509	20·8	608	116	19·1	401	55	13·7
8. 1.49	9,060	1,786	19·7	600	107	17·8	—	—	—
15. 1.49	9,030	1,753	19·4	890	143	16·1	—	—	—
22. 1.49	9,020	1,786	19·8	890	159	17·9	—	—	—
29. 1.49	9,000	1,698	18·9	890	161	18·1	—	—	—
5. 2.49	8,995	1,692	18·8	885	148	16·7	—	—	—
12. 2.49	8,980	1,827	20·4	870	144	16·6	—	—	—
19. 2.49	8,960	1,725	19·3	855	153	17·9	—	—	—
26. 2.49	8,955	1,809	20·2	855	182	21·3	—	—	—
5. 3.49	8,945	1,814	20·3	855	185	21·6	—	—	—
12. 3.49	8,940	1,857	20·7	855	177	20·7	—	—	—
19. 3.49	8,915	1,962	21·9	855	172	20·1	—	—	—
26. 3.49	8,865	1,903	21·5	855	158	18·6	—	—	—

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AN ANALYSIS OF ABSENCE UNDER A SCHEME OF PAID SICK LEAVE

BY

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In September, 1948, a scheme for paid sick leave was introduced for industrial workers in Government employment. By this scheme an employee who is absent from work because of illness receives his full flat rate of pay for as long as 13 weeks in any year. National Insurance benefit and any further allowances from other Government sources are deducted from this pay. All employees are entitled to receive sick pay once they have completed 26 weeks' Government service. If an employee has had five years' Government service, he is entitled to a further 13 weeks' absence on half pay. No one may receive more than one year's sick pay in any four years. Absences of three days only are not paid ; absences of four days receive one day's pay, but if a man is absent for a period of five days or more he receives sick pay for the whole period of absence. A doctor's certificate is required as evidence of incapacity.

After this scheme was introduced there was a sharp increase in sick absence. The increase was more than had been expected and was maintained into the year 1950. The scheme was to have been revised in September, 1950, but by June it was clear that too little was known about it, and in July, 1950, we were asked to make this investigation.

In 1949 the sick pay scheme applied to some 330,000 employees in 42 Government departments : 275,000 were employed in the four largest departments, the Admiralty, the War Office, the Air Ministry, and the Ministry of Supply. We decided to confine the study, initially at least, to these four departments, and visited their headquarters to discover what information was already available. Their statistics revealed that recorded sick absence as a whole appeared to have doubled. By far the larger proportion of that increase was due to longer rather than to shorter absences, particularly to absences of four weeks or more.

The increase of recorded sick absence was spread fairly evenly throughout the main disease groups.

The increase was real ; that is to say it was not to any extent accounted for by a reduction in absences of other kinds. (For example, there was a slight reduction in absences recorded as " casual absence ", as is to be expected when there is greater incentive to produce a medical certificate, but this reduction was negligible when taken in relation to the total increase in sick absence.)

There remained, however, several important questions which could not be answered by a study of headquarters statistics and made necessary a sample survey in industrial establishments. The questions we posed, and which are analysed in our results, were :—

(1) What proportion of the population records sick absence ? (2) How is the increase in sick absence distributed among people of different ages ? Is the present supposedly high rate of absence chiefly due to the employment of more elderly workers ? (3) The headquarters' statistics included long-term absences for which sick pay had been exhausted. To what extent do these long unpaid absences and the policy of Government establishments of retaining men on the books account for the present high rates ? (4) To what extent is the present rate due to the sick absence of the registered disabled ? (5) Do new entrants have a higher rate of sick absence than those of longer service ? (6) Are the sick absence rates of those in the more responsible and skilled jobs less than those of the unskilled worker ? (7) Is the rate for those on " incentive " pay (who revert to flat rates for sick pay) less than that of the time worker ? (8) Do workers take excessive sick absence before discharge or retirement ? (9) How does the sick absence of industrial workers compare with that of non-industrial workers whose sick pay scheme is of long standing ?

An exploratory study was first made to discover the best ways of collecting the information required,

and, after a standard method had been devised, 28 further establishments were visited between January 15 and March 29, 1951.

On each visit the method of collecting the information was explained and arrangements made for any modifications required for the particular establishment. When it appeared that the collection of any item of information might be difficult, owing to local variations in record keeping, we asked that it should be omitted rather than run the risk of inaccuracy. We asked for no information to be supplied except where we were satisfied, after inspecting the records, that it could be furnished accurately and without undue difficulty. On each visit the sick pay scheme was discussed with the executive and with workers' representatives.

Our information was collected by means of a standard form designed to contain a summary of an individual's sick absence from the period September, 1947, to August, 1950, and to contain other necessary facts such as his age, date of entering the establishment, type of work, wage, etc. Where an individual had entered after September, 1947, his sick absence record was taken from the date of entry. Wage, grade, and occupation were taken as at August 31, 1950.

In most cases the bulk of our information came from the wages record card. In some cases, where it was more convenient and we were satisfied of their accuracy, other sources were used. Supplementary information came from personnel records.

STANDARD FORM

Industrial "1" Non-industrial "2"		1
Name : SMITH, A. J.	Factory code	72
Address (Town) : Much Binding	Number 004321	
Enter in first space : Mr., Mrs. or Miss	Mr.	1
Date of birth (six figures)	26 12 01	
Date of entry Date of discharge (six figures). If not discharged, enter 000000	09 11 44 000000	
If registered disabled, enter 2. If not, enter 1		1
Trade in first space ..	Semi-skilled labourer	18
Basic wage, amount per week in shillings to nearest shilling		106
Bonus scheme, if any. Actual type in first space	Time rate	1

Details of sickness (and casual) absence from 9/47				Days lost : 9/47 to 8/48	017
				Number of absences : 9/47 to 8/48	1
Sick Absence			*Casual*		
1947 Oct.			1	Days lost : 9/48 to 8/49	056
Dec.			1		
				Number of absences : 9/48 to 8/49	4
1948 Feb.	14	Gastro-	Unpaid	Days lost : 9/49 to 8/50	015
Mar.	3	enteritis			
Sept.	5	Gastritis	Paid	Number of absences : 9/49 to 8/50	2
Oct.	1	Cold	,,		
Nov.	6	Not stated		Days lost six months before qualification	003
1949 Feb.	12	Bronchitis	,,		
Mar.	22	,,	,,	Days lost six months after qualification	024
Apr.	3	,,	,,		
				Discharges A. See notes	c
Aug.	7	Carbuncle	,,	Discharges B. See notes	
Sept.	9	,,	,,		
				Reason for discharge	
1950 Mar.	6	Bronchitis	,,		
Apr.			1		
July			1		

Sampling

It would have imposed too heavy a task to have asked for true samples of strengths of establishments in each of the three years we were studying. Our main sample was therefore taken from the men employed in the establishments at the time of our visit. In the tables given in this report it must be realized that the rates shown for the years September, 1947, to August, 1948, and September, 1948, to August, 1949, are not intended to represent the actual sick absence rates in these years ; they are the rates only for employees who remained in the establishments up to the time of our visits. In our tables, also, men are classified according to their ages in the year 1950 so that the rates given for the year 1947 to 1948 are for men who were two years younger at that time. In choosing establishments we aimed at a sample which would be representative of the four departments, and also of large and small establishments and of different geographical areas.

We wished to obtain sufficient numbers from the smaller establishments to enable comparisons to be made between establishments or groups of establishments. This usually involved asking for a sample of one in four of the total strength. At the larger establishments it appeared that the amount of work entailed in making returns for a sample of one in four would have been prohibitive, and the size of the samples was therefore reduced. This means that the larger establishments are under-represented in our sample, but we do not believe that this will bias the statistics contained in this report, unless it is later shown that the rates of sick absence in large and small establishments vary significantly. Samples were drawn from alphabetical or numerical lists of current employees. In all, some 8,000 records were included in our tabulations and, for most purposes, the number is sufficient. In the extreme age groups, however, the numbers are too few in some of our tables for comparisons at this age to be significant.

Sources of Error

Two operations were required to complete the standard form. The first was the direct copying of an individual's sick absence record and certain personal details from his wages sheet, sick pay card, or other sources. The second was the calculation of yearly totals of absence, and totals for six months before and six months after a man was eligible to receive sick pay. Each form was then examined and simple errors of calculation were corrected and then re-checked by another examiner. Wherever the errors were not obvious clerical slips and we could not make corrections ourselves with absolute confidence, the form was returned to the establishment for re-examination. To estimate the reliability of our own corrections, all doubtful forms from three establishments were returned for re-examination; where the errors were simple matters of calculation of such things as dates of qualifying for sick pay, our theoretical calculations of the date did not differ from the corrections made by the establishment by more than one or two days. When sick pay was shown as exhausted but the date did not tally with our own calculations, the record was again suspect and returned for re-examination. This method of checking brought to light some forms where there had been errors in the recorded details of sickness as well as in the calculation of totals, and it must be assumed that there were other forms in which details had not been recorded accurately. However, from the number of forms which had to be returned to any one establishment, we have a crude indication of the probable accuracy of the clerical work done there and of the probable accuracy of our returns.

We believe that the hidden errors of copying would be largely errors of omission. It would be possible to omit an instance of recorded sickness but unlikely that additional instances of sickness would be invented, especially where the diagnosis had also to be entered. In establishments where the clerical work did not seem to have been very accurate, it is possible that the overall figures of absence given us would be lower than the true ones. This would become important when comparing one establishment with another. But it seems likely that such omissions would be random, and that the differences between the large groups which form the basis of this report would be unaffected. The same conclusions apply to other types of error in the forms themselves.

Another possibility of error arises with the transfer of the information from our form to the Hollerith punch card. Again we have a check only when the error results in an apparent impossibility. After sorting we had, for instance, 15 cards with ages recorded as from 1 to 10 years or from 90 to 100. This is a small number in some 8,000, but indicates the probability of other errors of punching. But again there is no reason to suppose that such errors are not distributed at random and, with the large numbers involved, our comparisons should remain unaffected.

We conclude that our data are reliable for the purposes to which they have been put.

Results

This report is confined to the male population which forms the large majority of the total.

The average number of days lost per employee is the most convenient way of expressing sick absence rates, but it can be very misleading when used in comparing small groups of people. In such cases, the chance inclusion or exclusion of even one or two people with very long absences will greatly alter the average rates. In the tables we have also given the average length of absence for each occasion of sickness, the number of occasions of sickness for every 100 employees, and the percentage of the population which records any sick absence in the periods under consideration. All tables refer to working days lost.

(1) What Proportion of the Population Records Sick Absence? Table 1 shows that the percentage of the population with one or more occasions of sick absence in the year increased from 27 in 1947–48 to 46 in 1949–50. It is worth noting that over half the population still recorded no sick absence in the latter year.

(2) How Is the Increase Distributed Among Different Ages? Table 1 also shows that in both 1947–48 and in 1949–50 the average number of days lost rises with age. But the increase during 1948–50 is relatively the same for each age group, except for the men under 30 for whom the increase is less. Among the men under 25 there has been a greater increase in the number who record sickness absence, but very few of this age group recorded sickness absence before paid sick leave was introduced.

The absences for men of 56 and over have been extracted from the total, and the average number of days lost for the remainder is shown. The overall average is then reduced from 13·4 to 11·7 in 1948–49 and from 14·1 to 12·5 in 1949–50. Although the average absence of the older men is high, the numbers involved are not sufficient to alter the general average to any great extent. It cannot be said that the present rates are to any major extent due to the more elderly employees.

TABLE 1

SICK ABSENCE OF TOTAL SAMPLE (MALE INDUSTRIALS)

Age at 1950	September, 1947–August, 1948					September, 1948–August, 1949					September, 1949–August, 1950				
	No.	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentage with Sickness	No.	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentage with Sickness	No.	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentage with Sickness
61 and over	579	6·4	14·2	44·9	31	606	16·6	24·3	68·0	47	629	17·8	25·0	71·2	49
56–60	735	6·9	13·3	52·1	32	763	18·7	24·3	76·8	51	796	19·5	25·2	77·5	51
51–55	866	5·6	12·0	46·9	27	891	16·0	23·8	67·3	45	927	15·7	22·2	70·8	48
46–50	693	5·0	13·6	36·6	25	715	13·2	21·6	61·3	42	760	13·3	20·7	64·1	44
41–45	626	4·3	12·2	35·1	24	651	10·6	17·1	61·9	42	704	11·9	19·3	61·8	43
36–40	548	3·2	9·0	35·8	22	574	9·5	16·1	58·7	40	625	10·7	16·2	65·9	42
31–35	301	4·4	10·3	42·5	30	341	7·9	14·1	56·0	39	383	12·9	18·7	68·9	45
26–30	242	4·6	12·0	38·0	25	291	10·0	14·2	70·5	45	373	10·4	13·9	75·0	44
21–25	105	4·8	15·1	31·4	17	163	7·5	13·0	57·7	40	236	9·9	16·4	60·6	42
20 and under	51	(3·9)	(7·4)	(52·9)	(25)	74	(6·3)	(10·1)	(62·2)	(38)	101	(4·8)	(8·4)	(57·4)	(41)
	4,746	5·2	12·4	42·1	27	5,069	13·4	20·4	65·3	44	5,534	14·1	20·6	68·7	46

Average days lost, excluding men of 56 and over = 11·7 ..... = 12·5

PERCENTAGE INCREASE OF 1949–50 OVER 1947–48

	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentage with Sickness
61 and over	178	76	59	58
56–60	183	89	48	50
51–55	180	85	51	78
46–50	166	52	76	76
41–45	177	58	76	79
36–40	234	80	84	91
31–35	189	82	62	50
26–30	126	16	97	76
21–25	(106)	(9)	(96)	(147)
	—	—	—	—
	171	66	63	77

(3) To what Extent Does the Inclusion of Long Term (Unpaid) Sick Absence Account for Apparent High Rates? For all but five establishments our records show the proportion of long absences which are unpaid or at half pay. Table 2 shows the average rates before and after both unpaid and half-paid absence is extracted. In the year 1949–50 the average number of days lost is reduced from 14·6 to 11·9. (This reduced rate cannot be compared with the rate for 1947–48, since it is not possible in that year to make a reliable estimate of the absence which would represent unpaid absence under the scheme.) But even after the extraction of unpaid absence, the rate of absence appears formidable compared with that for 1947–48.

(4) To What Extent Is the Present Rate due to the Sick Absence of the Registered Disabled?—The standard form on which information from establishments was gathered contained a space for entering whether the individual was registered disabled or not. The number of registered disabled represented approximately 7·2% of the total for whom this entry was properly completed. The relative rates of sick absence are shown by the following figures.

	No.	Sept., 1947–Aug., 1948	No.	Sept., 1949–Aug, 1950
		Average No. Days Lost		Average No. Days Lost
Recorded as registered disabled	289	9·1	332	25·3
Recorded as not registered disabled	3,889	5·1	4,582	13·7
Total population ..	4,746	5·2	5,534	14·1

Although the number of registered disabled is too small for exact comparison, two observations can be made. While the rate for the group as a whole is high compared with the rest of the population, the proportion of registered disabled is so low that their exclusion does not have much effect on the overall rate. The rate for those recorded as not registered disabled is only 0·4 days less than that of the total population in the period 1949–50. Moreover, the registered disabled had higher rates before the scheme was introduced and there is very little difference in the percentage increase on the year before the scheme. It cannot be said that the sick absence of the registered disabled has had any great effect on the present overall rates.

TABLE 2
ABSENCE AT HALF AND NO PAY

Age at 1950	September, 1949, to August, 1950		
	No.	Average Days Lost	Average Days Lost after Extraction of Absences on Half and No Pay
61 and over ..	489	18·5	15·3
56–60.. ..	634	19·7	14·7
51–55.. ..	713	15·8	12·7
46–50.. ..	592	13·4	11·1
41–45.. ..	531	13·5	10·6
36–40.. ..	459	10·3	8·5
31–35.. ..	295	13·2	12·2
26–30.. ..	256	10·5	9·9
21–25.. ..	182	11·5	3·7
20 and under	62	4·5	4·4
Total ..	4,213	14·6	11·9

(5) Do New Entrants Have a Higher Rate than Men of Longer Service?—Men who entered after the scheme was introduced had only six months during which they were unable to draw sick pay. We have therefore calculated the rates during the six months before and the six months after they were qualified for sick pay. Rates for six months before and after qualification have also been calculated for men of longer service. With the men of longer service the six months before qualification is the period March to August, 1948, and the six months after qualification is the period September, 1948, to February, 1949; for the new entrants these periods before and after qualification occur at varying times of the year. For the men of longer service we are always comparing a winter with a summer rate. One would expect, other things being equal, that the group of men with longer service would show a higher rate in the six months after qualification and a larger increase over the six months before. Table 3 gives the position. While the increase is certainly greater in men with longer service, the rates for the middle age groups of the new entrants is higher in the six months after qualification than that of the men with longer service. However, due to the disparity in age distribution, the overall rates for the two groups are much the same.

Without estimating the rates to be expected at different times of the year, it is impossible to state definitely whether or not new entrants have relatively greater sick absence than men of longer service, but the figures suggest that any difference would be small. From this and from the fact that the new entrants represent only a small proportion of the total population, it can be concluded that it would not be worth paying any special attention to this question. There is certainly no evidence on these data to support one of the suggestions made to us that the period of qualifying service should be increased. That would have little effect beyond reducing the total numbers of men eligible for sick pay. It must nevertheless be pointed out that our survey was completed before recruitment under the rearmament programme had properly begun, and, where there has since been a large influx of new labour, possibly recruited at a time when labour was scarce, the above conclusion may not hold true.

(6) Are the Rates of More Responsible and Skilled Workers Less than the Unskilled?—The population

TABLE 3
SICK ABSENCE AND LENGTH OF SERVICE

Age at 1950	Long Service							New Entrants						
	Six Months before Qualification			Six Months after Qualification				Six Months before Qualification			Six Months after Qualification			
	No.	Average No. Days Lost	Percentage with Sickness	No.	Average No. Days Lost	Percentage with Sickness	Percentage Increase	No.	Average No. Days Lost	Percentage with Sickness	No.	Average No. Days Lost	Percentage with Sickness	Percentage Increase
61 and over	569	3·1	18	569	8·6	33	177	57	5·0	22	50	8·5	35	70
56–60 ..	708			708				71			63			
51–55 ..	847	2·6	16	847	7·2	28	177	71	4·1	17	60	8·8	35	115
46–50 ..	673			673				84			66			
41–45 ..	624	1·9	12	624	4·8	28	153	96	3·5	19	76	6·6	34	89
36–40 ..	545			545				95			75			
31–35 ..	300	1·8	15	300	4·7	27	161	102	2·5	18	75	6·2	33	148
26–30 ..	242			242				158			130			
21–25 ..	107	2·5	15	107	4·6	30	84	159	1·8	14	129	3·8	28	111
20 and under	51			51				91			50			
	4,666	2·5	15	4,666	6·6	29	164	984	3·1	18	774	6·5	33	110

Overall Totals
Six months before qualification (5,650 workers) average days lost = 2·6; yearly equivalent = 5·2.
Six months after qualification (5,440 workers) average days lost = 6·6; yearly equivalent = 13·2.

was divided into three main trade groups, skilled, unskilled, and miscellaneous :—

Trade Group I.—This comprises boiler-makers, borers, carpenters, electricians, examiners, fitters, instrument makers, joiners, markers-out, millers, millwrights, setters, shipwrights, ship-fitters, pattern makers, tool and gauge makers, turners, and armourers. These are classed as skilled trades in all four departments.

Trade Group II.—This comprises brush hands, clothing workers, crane drivers, fire brigade men, firemen, labourers (skilled and unskilled), mates, machine hands, M.T. drivers, packers, packing case makers, process workers, slingers, stokers, storemen, and truck drivers. These are classed as unskilled trades.

Trade Group III.—This comprises chasers, inspectors, painters, viewers, and leading hands (whom it is apparently difficult to class as invariably skilled or unskilled), and various miscellaneous trades which, because of their infrequency, were not given separate code numbers on the punch cards. Incomplete forms in which the trade was not given are also grouped here.

Overlookers, leading storemen, and telephone operators were excluded from these groups because men in these trades were entitled to some sick pay before September, 1948. Apprentices and cleaners were also excluded, apprentices because their sick rates were peculiarly low, and cleaners because among them are found many part-time workers.

Table 4 gives the rates for Trade Groups I, II, and III.

TABLE 4

ANALYSIS OF SICKNESS ABSENCE RELATED TO SKILLED AND UNSKILLED TRADE GROUPS

Age at 1950	September, 1947–August, 1948					September, 1949–August, 1950					Percentage Increase			
	No.	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentage with Sickness	No.	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentage with Sickness	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentage with Sickness
Trade Group I : Skilled														
61 and over	68	6·8	13·4	51·2	28	72	17·6	25·0	70·5	46	159	87	38	64
56–60	91					100								
51–55	123	5·2	16·0	32·6	23	133	13·1	21·5	60·9	45	152	34	87	96
46–50	135					146								
41–45	150	3·2	11·2	29·6	21	167	9·8	15·3	64·4	45	206	37	118	114
36–40	144					164								
31–35	85	3·5	10·1	34·8	24	104	9·2	14·6	63·3	42	163	45	82	75
26–30	96					141								
21–25	49	(2·6)	(7·8)	(34)	(21)	91	(8·3)	(13·5)	(61·2)	(41)	—	—	—	—
20 and under	4					7								
	945	4·2	12·7	33·1	23	1,125	11·6	18·1	63·9	44	176	43	93	91
Trade Group II : Unskilled														
61 and over	370	6·8	13·4	51·2	32	404	19·8	24·8	79·9	51	191	85	56	59
56–60	458					498								
51–55	514	5·7	13·0	44·1	28	547	15·8	21·9	72·1	48	177	68	63	71
46–50	375					412								
41–45	323	4·2	11·0	38·1	24	356	13·2	19·8	66·7	44	214	80	75	83
36–40	263					287								
31–35	140	6·0	14·6	41·1	26	169	11·0	14·0	78·6	44	83	0	87	67
26–30	101					139								
21–25	32	(3·9)	(7·5)	(52·8)	22	86	(10·6)	(20·1)	(52·7)	(39)	—	—	—	—
20 and under	4					7								
	2,580	5·7	12·8	44·9	28	2,905	15·8	21·5	73·4	47	177	68	63	68
Trade Group III : Miscellaneous														
61 and over	115	7·2	20·2	35·6	—	126	16·6	26·7	62·3	—	131	32	75	—
56–60	135					147								
51–55	170	3·6	12·6	28·7	—	187	12·5	20·8	60·0	—	253	65	109	—
46–50	130					148								
41–45	126	2·7	10·9	24·9	—	154	9·3	15·8	58·9	—	244	45	137	—
36–40	115					148								
31–35	66	2·6	6·9	38·1	—	99	11·0	15·3	72·5	—	323	122	117	—
26–30	44					90								
21–25	16	(3·3)	(9·6)	(34·8)	-	45	10·2	14·2	71·9	—	(239)	(48)	(107)	—
20 and under	7					12								
	924	4·2	13·7	30·7	—	1,156	12·3	19·6	62·7	—	193	43	104	—

It will be seen that the present rates for the unskilled trades are higher than those for the skilled trades. Yet the rates for the unskilled workers for the year before the scheme was introduced were also higher. There is little difference in the proportional increase. There is a lower increase among the more elderly in the skilled trades but a higher increase among the younger people. The unskilled show a smaller increase in the number of occasions of absence and a larger increase in the duration of absences. These are crude estimates but support the assumption that the unskilled groups contain a larger number of relatively unfit men. They do contain a higher proportion of registered disabled, and the extraction of these from both groups reduces the disparity to some extent. It may be assumed that they also contain a higher proportion of men who are relatively unfit but who do not choose to register as disabled persons.

The 1949–50 rates for Trade Group III (miscellaneous) are similar to those of the skilled trades. The greater increase shown for the middle age groups, due to lower rates in 1947–48, is of doubtful significance but has not yet been investigated.

It may be concluded that, although the unskilled workers have rather higher absence rates than the rest of the population, the proportional increase of the present rates over those before the sick pay scheme was introduced is no different. There is no evidence to suggest that anything would be gained by paying special attention to this group of workers.

(7) Is the Absence for Workers on "Incentive" Pay Less than that of the Time-worker?—The population was divided into those who were paid on a flat rate or time rate basis and those who received some form of incentive payment. The incentive group included all who received an incentive bonus, piece work rates, group bonus, works bonus, joint contract, and those who varied but were predominantly on one or other of these incentive types of payment. Table 5 gives the absence rates for these two groups.

It will be seen that the flat rate workers have a markedly higher sick absence rate at all ages; they have a higher proportion of people recording sick absence, a higher number of absences, and a higher average length of absence. The proportional increase in the average number of days lost is

TABLE 5

ANALYSIS OF SICKNESS ABSENCE RELATED TO THE METHOD OF PAYMENT

Age at 1950	September, 1947–August, 1948					September, 1948–August, 1949					September, 1949–August, 1950					Percentage Increase (Average Days Lost)
	No.	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentage with Sickness	No.	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentage with Sickness	No.	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentage with Sickness	
Flat Rates																
61 and over ..	445	6·6	14·0	47·0	31	471	17·1	24·3	70·3	48	489	18·3	25·8	70·8	48	177
56–60	492	7·5	12·9	58·1	34	517	20·6	25·4	81·2	53	546	21·8	26·0	84·1	52	191
51–55	557	5·9	11·1	52·6	29	579	18·0	25·1	71·9	46	612	16·7	22·2	75·2	50	183
46–50	383	5·6	12·8	43·6	28	401	15·3	22·8	67·3	46	438	15·4	21·3	72·2	47	175
41–45	382	4·7	12·7	36·9	24	399	12·1	19·1	63·2	44	441	13·7	20·8	65·8	44	191
36–40	342	3·6	9·3	39·2	23	362	11·0	18·0	60·8	41	406	12·4	17·6	70·7	44	244
31–35	202	4·0	9·8	40·6	29	233	8·9	15·1	59·2	40	265	14·3	20·6	69·4	47	258
26–30	135					172					243					
21–25	55	5·3	17·1	31·2	19	102	9·4	14·0	67·2	43	160	10·8	15·1	71·2	43	104
20 and under..	28					46					72					
	3,021	5·6	12·3	45·7	28	3,282	15·0	21·7	68·9	46	3,672	15·7	21·6	73·0	47	180
Incentive Rates																
61 and over ..	124	5·6	14·5	38·7	28	125	15·0	24·4	61·6	44	130	15·7	21·5	73·1	51	180
56–60	217	6·0	14·4	41·9	29	220	14·4	21·6	66·4	44	224	13·6	22·7	59·8	42	127
51–55	290	5·3	14·5	36·2	27	293	12·3	21·5	57·3	41	296	13·9	22·9	60·8	45	162
46–50	290	4·2	14·7	28·6	22	294	10·3	19·0	54·1	37	302	9·4	18·0	52·3	39	124
41–45	242	3·6	11·2	32·2	23	250	8·2	13·6	60·0	40	261	8·9	16·3	54·8	41	147
36–40	203	2·6	8·4	30·5	19	209	7·0	12·6	55·5	38	216	7·5	13·0	57·9	37	188
31–35	98	(5·3)	(11·2)	(46·9)	(32)	107	(5·9)	(11·9)	(49·5)	(38)	117	(9·9)	(14·7)	(67·5)	(42)	(87)
26–30	107					119					130					
21–25	50	3·6	7·7	46·7	27	61	7·7	12·4	62·5	42	76	6·8	11·2	60·8	43	89
20 and under ..	23					28					29					
	1,644	4·5	12·2	36·3	25	1,706	10·2	17·4	58·6	40	1,781	10·5	17·8	59·3	42	133

greater at all ages except those of 61 and over where it is much the same.

The population was further divided to compare the effect of flat rate and incentive pay within the groups of skilled and unskilled workers (Table 6). The difference remains distinct and is most marked in the average length of absence. A separate analysis showed the incentive workers to have a higher proportion of absences from one to 10 days and the flat rate workers to have a higher proportion of absences from 11 to 40 days, the greatest difference occurring in absences from 11 to 25 days.

It is interesting to note that the difference between flat-rate and incentive workers is maintained among the new entrants, although the numbers are too small to be reliable.

New entrants on flat rates :

Six months before qualification .. average days lost 3.15 (803 workers)

,, ,, after ,, .. average days lost 6·76 (638 workers)

Increase = 115%.

New entrants on incentive payment :

Six months before qualification .. average days lost 2·76 (181 workers)

,, ,, after ,, .. average days lost 5·15 (136 workers)

Increase = 87%.

TABLE 6

TRADES AND METHOD OF PAYMENT IN RELATION TO SICK ABSENCE RATES

Age at 1950	September, 1947–August, 1948					September, 1949–August, 1950				
	No.	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentage with Sickness	No.	Average Days Lost	Average Length of Absence	Absences per 100 Workers	Percentages with Sickness
Unskilled Flat Rate										
56 and over	647	7·2	13·6	53·2	33	716	21·3	25·6	83·4	55
46–55..	584	6·1	12·1	50·4	30	644	17·3	22·3	77·8	51
36–45..	401	4·2	11·8	35·7	23	449	15·0	20·8	71·9	47
26–35..	175	5·7	13·4	42·4	25	229	12·3	16·7	73·4	41
25 and under ..	28					75				
	1,835	6·0	12·8	47·2	29	2,113	17·4	22·4	77·8	49
Unskilled Incentive										
56 and over	181	5·5	12·5	44·2	30	186	14·2	21·3	66·7	45
46–55..	305	5·1	15·9	32·1	24	315	12·7	20·9	60·6	43
36–45..	185	4·2	9·5	43·8	29	194	9·2	16·8	54·6	36
26–35..	66	(5·9)	(13·6)	(43·2)	(28)	79	(6·8)	(9·6)	(70·1)	(42)
25 and under ..	8					18				
	745	5·1	12·9	39·1	26	792	11·4	18·6	61·7	42
Skilled Flat Rate										
56 and over	77	(5·0)	(14·7)	(33·8)	(25)	86	(19·5)	(28·6)	(67·4)	(43)
46–55..	108	5·2	20·2	25·9	20	122	16·7	26·5	63·1	46
36–45..	132	3·4	11·1	30·3	20	154	12·1	17·2	70·8	51
26–35..	83	2·9	15·3	18·8	15	126	8·5	14·4	59·4	41
25 and under ..	18					49				
	418	4·02	14·9	27·1	20	537	13·2	20·4	65·2	44
Skilled Incentive										
56 and over	82	6·2	14·5	42·7	30	86	15·7	21·4	73·3	49
46–55..	150	5·2	13·9	37·3	25	157	10·3	17·4	59·2	45
36–45..	162	3·3	11·3	29·0	22	177	7·8	13·2	58·8	46
26–35..	98	3·7	7·8	46·6	30	119	9·4	14·2	66·1	43
25 and under ..	35					49				
	527	4·4	11·5	38·0	26	588	10·1	15·9	63·1	45

Since these types of incentive payment are made only in nine of the 25 establishments studied, it would be possible for the difference between sick absence rates to be due to the establishments themselves. However, a further analysis was made and revealed that in those establishments with workers both on time and incentive pay, the time-paid workers had a similar sick absence rate to the group of time-paid workers as a whole.

It would seem that a distinct difference can be established between these two groups of workers both in their present rates and in the increase over the rates before the sick pay scheme was introduced. It is highly probable that the difference is due to the fact that the worker who receives incentive payment reverts to his flat or basic rate of pay when sick ; he gets more money when at work. The worker on flat rates of pay gets the same money

whether at work or sick; in many instances he may be better off financially when he is absent sick, apart from any expenses caused by the illness itself. No attempt was made to calculate the relative differences in payments received at work or when sick in these two groups.

It must not however be assumed that the difference between the present sick absence of workers on flat rate and incentive rate is all due to such financial causes. Flat-rate workers include a higher proportion of registered disabled and had higher rates before the scheme. The important difference is the consistently higher increase. For example, if the 1947–48 sick absence of the flat-rate workers is increased by the proportion shown for incentive workers the overall sick absence rate would be about 13·4. Nevertheless, flat-rate workers form the majority of the population, and even a minimum reduction of about two days in an average of 15·7 might still be worth attention.

(8) Do People Take Excessive Sick Absence Just before Leaving?—We collected representative samples of men who had left the establishments during the year September, 1949, to August, 1950. Since a large number of these were men with short periods of service, we compared the average rate of absence for six months before qualification with the rate for a period up to six months in all, for which they were qualified just before leaving.

Many men were not qualified for as long a period as six months before they left, but we were able to calculate the average rate for the group as a whole (and for each separate age group) according to the following method :—

We calculated the number of days during which a man was qualified for sick pay within six months (or 130 days) of leaving and we recorded the number of days sick absence taken in this period. If Q is the sum of the days for which each member of a group was qualified within six months of leaving, and A is the sum of the days lost in this period, then A multiplied by 130 and the result divided by Q is the average number of days lost by the group as a whole for six months of qualified service before leaving.

Table 7 shows this comparison. The figures are given for those who left for the following official reasons :—" Resigned " (also " voluntarily retired " if obviously not for age reasons), " domestic ", " marriage ", " other work ", " leaving district ", " dissatisfied ", " disgruntled ", " travel difficulties ", " redundant ", " termination of temporary employment ", " unsuitable ", " no suitable work available ", and some other odd reasons. We have not included transfers from one establishment to another, nor have we included those who are stated to have left for medical reasons.

Table 7 clearly shows a disproportionate amount of sick absence in the period immediately before leaving and a disproportionate increase over the rates

TABLE 7

ANALYSIS OF ALL LEAVERS EXCEPT ON MEDICAL GROUNDS AND TRANSFERS

Age at 1950	Six Months before Qualification				After Qualification and just before Leaving			
	No.	No. Days Lost	Average No. Days Lost	Half-yearly Rate 1947–1948	Q. No. Days Qualified up to 130 Days before Leaving	Average No. Days Lost at Q.	Average No. Days Lost $\frac{Ax130}{Q}$	Half-yearly Rates 1949–1950
61 and over	111	426	3·7	3·3	14,279	2,360	20·7	9·0
56–60	40	139			5,077	801		
51–55	78	285	2·7	2·7	10,011	1,606	12·8	7·1
46–50	84	158			10,218	956		
41–45	92	253	3·0	1·9	10,759	732	9·5	5·7
36–40	106	341			12,649	985		
31–35	103	176	1·8	2·2	12,060	1,209	11·3	5·8
26–30	140	261			16,605	1,125		
21–25	101	217	2·1	2·2	10,542	536	6·5	4·2
20 and under	10	115			1,102	47		
	863	2,371	2·74	2·6	103,302	9,857	12·3	7·1
					Redundant Only			
61 and over	76	335			9,750	1,674		
56–60	16	75			2,080	496		
51–55	32	173			4,160	507		
46–50	36	59			4,261	525		
41–45	30	72	3·3	2·6	3,497	168	16·4	7·1
36–40	31	180			3,414	307		
31–35	25	31			3,024	459		
26–30	33	23			3,810	219		
21–25	12	9			1,065	73		
20 and under	1	0			130	0		
	292	957			35,191	4,428		

in the six months before qualification. These rates may be compared with half the average rates shown for the whole population in 1949–50. This finding must be interpreted with due caution. The figures do not prove that any of this absence is excessive in the sense of being "unnecessary". Many of these men may have left the establishment for medical reasons, although this may not have been recorded officially among the reasons for leaving. This is particularly likely to be the case among those recorded as having "retired" or "resigned". Others may have decided to leave because recent ill-health had made them wish to change their work, even though it could hardly be said that they had left because they were no longer fit for the work they had been doing. A further group of 136 men, marked simply as "retired", shows an increase from 3·8 days for the six months before qualification to 20·4 days for the six months before retirement. They are all over 61 years old.

Table 7 also shows the rates for men discharged as redundant and, though the numbers are small, a similar disproportionate increase is shown. While this more strongly suggests that men take unnecessary absence before leaving, it is again not proved; it is possible that there is a tendency when declaring men redundant to choose first those who have an unsatisfactory attendance record.

There is certainly a disproportion of sick absence recorded for men about to leave.* It is possible, even likely, that some of this can be regarded as medically unnecessary, but we cannot give a positive statement about it without special study of the detailed records and supplementary studies of sample cases. As the number of leavers is few in relation to total establishment strengths, this problem would only become serious in the event of some future large scale reduction in the number of people employed in Government establishments.

(9) How Does the Sick Absence of Industrial Workers Compare with that of Non-industrial Workers?—Table 8A gives the sick absences for non-industrial workers during the three years from September, 1947, to August, 1950. In the year 1947–48 the sick absence rates of non-industrial workers exceeded those of the industrial workers at all ages; in the next two years the position is reversed. There has, nevertheless, been an increase in the rates for non-industrial workers which is consistent in that it occurs at all ages. This increase may be due to the introduction of the National Health Insurance Scheme in 1948 but, whatever the cause, it is likely to be one which is common to both industrial and non-industrial workers. It is probable that part of the increase in industrial sick absence can be ascribed to other causes than the introduction of the sick pay scheme alone.

The present rates for the industrial population as a whole are higher than those of the non-industrial population, but reference to Table 6 shows that the rates for industrial workers on incentive payment are about the same as those for the non-industrial workers. The figures for the non-industrial workers include the short uncertified absences to which they are entitled; there is probably no difference between the cost of non-industrial sick absence and that of the industrial

TABLE 8A

SICK ABSENCE IN NON-INDUSTRIAL WORKERS (TOTAL SAMPLE)

Age at 1950	September, 1947–August, 1948					September, 1948–August, 1949					September, 1949–August, 1950				
	No.	Average Days Lost	Average Length of Absence	Absence per 100 Workers	Percentage with Sickness	No.	Average Days Lost	Average Length of Absence	Absence per 100 Workers	Percentage with Sickness	No.	Average Days Lost	Average Length of Absence	Absence per 100 Workers	Percentage with Sickness
61 and over	156	11·0	7·1	164·2	64	166	12·1	7·7	156·6	66	168	13·2	8·5	155·9	65
56–60 ..	239	9·6	6·8	140·0	61	245	13·5	9·1	148·6	62	252	14·1	8·8	161·5	68
51–55 ..	364	8·8	6·3	138·5	63	377	10·2	7·0	146·4	67	388	12·3	7·4	165·2	70
46–50 ..	310	7·2	5·2	138·0	62	320	7·6	4·8	158·8	67	331	8·2	5·7	144·7	61
41–45 ..	289	6·1	4·6	132·2	59	302	8·1	5·6	144·4	63	319	8·8	6·1	142·6	66
36–40 ..	216	8·0	4·8	167·6	69	223	8·7	5·1	172·2	70	233	8·6	5·3	163·1	67
31–35 ..	131	4·9	3·0	161·8	64	137	8·4	5·0	167·2	65	152	7·4	4·4	169·1	68
26–30 ..	95					103					123				
21–25 ..	25	4·4	3·0	146·3	58	30	6·7	4·0	168·7	67	37	7·7	4·5	169·3	71
20 and under	1					1					3				
	1,826	7·8	5·3	145·4	62	1,904	9·5	6·1	155·4	66	2,006	10·2	6·5	157·4	67

* Our visits were paid between September, 1950, and March, 1951. In collecting our samples from the present strengths of establishments we will have included some men who would have been near to leaving in the year 1949–50; this may have slightly exaggerated the rates shown in our tables for this year.

TABLE 8B

CERTIFIED AND UNCERTIFIED ABSENCE (REDUCED TOTAL)

Age at 1950	September, 1947–August, 1948				September, 1949–Augus., 1950			
	No.	Average Days Lost Certified	Average Days Lost Uncertified	Percentage with Uncertified Absence	No.	Average Days Lost Certified	Average Days Lost Uncertified	Percentage with Uncertified Absence
61 and over ..	127	11·02	1·42	51	138	11·49	1·08	42
56–60.. ..	178	9·03	1·28	53	189	12·85	1·48	52
51–55.. ..	290	7·86	1·28	52	307	10·39	1·36	53
46–50.. ..	258	5·84	1·25	49	274	6·73	1·27	48
41–45.. ..	230	5·17	1·23	46	255	7·51	1·22	49
36–40.. ..	165	5·13	1·58	61	176	6·85	1·32	52
31–35.. ..	100	3·75	1·46	56	111	5·54	1·50	55
26–30.. ..	70				85			
21–25.. ..	20	3·12	1·34	52	30	7·42	1·53	62
20 and under..	1				37			
	1,439	6·60	1·33	52	1,568	8·70	1·33	51

workers on incentive payment. (The sample of non-industrial workers includes men in higher executive posts.)

There are, however, considerable differences in the ways in which sick absence is taken. As would be expected, the non-industrial worker takes more frequent short absences and fewer people take no absence at all. Table 8B gives the rates for certified and uncertified sick absence separately. It will be seen that there has been no increase in the average number of days lost from uncertified sickness and no increase in the proportion of people who record uncertified sick absence. Consequently the increase in non-industrial sick absence must have been due to certified sick absence alone.

These figures suggest that we should look for administrative causes following the introduction of the National Health Service as an explanation of at least part of the increase among non-industrial workers and as an explanation of some part of the increase among industrial workers.

Discussion

We have attempted to get a clearer statistical picture of the sick absence rates and the way in which they have increased after the introduction of the sick pay scheme. We have compared rates between different groups of workers and we have estimated the relative importance of the contribution made by some groups towards the general rate of sick absence. Our chief object has been to discover whether there are any groups of people who contribute more than others to the general increase and whether the total contribution is sufficient to make it worth taking practical steps to remedy the situation.

By this method it has been possible to investigate some of the questions posed by the increase in sick absence without having to resort to comparisons with other industrial organizations. But it should be noted that similar rates of sick absence have been reported by other organizations which have sick pay schemes. We have examined statistics for one factory belonging to a private organization. At this factory half the population were regarded as established workers and received sick benefit on terms similar to those received by Government industrial workers. The sick absence rates for those who received benefit were strikingly higher than the rates for those who did not. The sick benefit scheme had been in operation for many years, and it was shown that the increase took place as soon as men became eligible for sick pay. The difference between the rates of those who did and did not receive sick benefit was of the same order as the difference between the 1947 and 1950 rates at a nearby Government factory which was doing similar work. We think it is unlikely that the present sick absence rates will decline to any marked extent as time goes on. We have found no evidence to support a suggestion that the present sick absence rates are to any extent caused by some deep-seated state of psychological unrest among the working population at the present time.

The increase in sick absence among non-industrial workers can perhaps be explained by the introduction of the National Health Service for the whole country in 1948. If this were so, the increase among industrial workers could presumably be ascribed in part to this cause as well as to the introduction of the sick pay scheme.

We find no evidence that a sufficient explanation of the general increase is to be found in terms of the age distribution of Government industrial workers, nor that the proportion of registered disabled workers accounts for more than a small fraction of the present rate. We have extracted the proportion of unpaid long-term absence, and

find that the rate for paid absence still appears formidable compared with the overall rate before the scheme was introduced. There is little evidence that new entrants (in the three years covered by our survey) have contributed any undue amount to the absence rate. We find insufficient difference between the broad classification of skilled and unskilled workers to concentrate the search for remedies on the one class or on the other. There is a high rate of sick absence among those who are about to leave although it is not possible to explain it fully. But the magnitude of this problem is dependent on labour turnover ; it would only be worth attention in the event of some future large scale reduction in the number of men employed.

We do find a distinct and striking difference between workers who receive incentive payment and those who do not. It is possible that part of this difference might arise from selection, the less fit workers gravitating to jobs involving less speed. However, not only the sick rate but the proportional increase in days lost after the introduction of the scheme is lower for the incentive worker, and this suggests that the smaller absence rate of the latter is in part due to the fact that he receives less money when absent sick than when at work. Yet it is unlikely that he is driven to work by dire necessity. We do not believe that this earlier return to work has any ill effects on health, although investigation of this point was beyond the scope of this stage of our enquiry. It is likely, for reasons which will be discussed later, that it may have some beneficial effects on health.

For the time worker the monetary incentive may work the other way round. He receives his full pay when he is absent sick, and he has no travelling expenses ; if he pays income tax, the exemption of that part of his wage which is National Health Insurance may take him out of the tax-paying class ; in some instances he may draw extra benefit from his own sick club or clubs.

The introduction of any sick pay scheme of this kind will enable many people to be absent who ought to have been absent before. Many executive officials stressed this aspect to us, and commented on the number of cases before the scheme where men came to work who ought to have stayed at home. To this extent some increase in sick absence was to have been expected, although it is impossible to estimate how much. But one would expect the increase in " necessary " absence to bear some constant relation to sick absence rates before the scheme was introduced. Where it is found that one group of workers has both a higher rate and a larger proportional increase than another, and where no explanation can be found in terms of age, conditions of work or other causes, we presume, tentatively, that there is unnecessary absence in the group with the higher rate. We use the term " unnecessary absence " deliberately because the adjective implies no condemnation.

With this sick pay scheme a man may remain absent from work until he and his doctor decide that he is fully recovered from an illness. If there is a reasonable incentive to return to work he may well decide that he is fit enough to do so at a relatively early stage. But, if there is no such incentive to return to work, he may both feel and be unwell for a longer time, and many people will display the signs of illness. The patient may be totally unaware of the connexion between his continued ill-health and the lack of an incentive to get well. No amount of exhortation will convince him that his absence is unnecessary.

This raises an important medical aspect of the problem of paid sick absence. When there was a strong financial incentive to remain at work or to return to work as soon as possible, the doctor had the problem of persuading, even " ordering " a sick man to stay at home. In the absence of such an incentive he may have to consider whether a patient should be persuaded to return to work in order to hasten his recovery. To some extent this problem represents a change of attitude on the part of the doctor ; he may be aware of it but, in the ordinary way, he has seldom had to take action about it. This adds a medical argument to the need for some tangible incentive to early recovery.

We heard a good deal in the course of our visits about the problem of creating the right morale. Undoubtedly morale affects sick absence rates. But the comments we heard generally implied that an attitude of hostility should be built up towards the suspected malingerer. Although in many cases the judgment of fellow workers against suspected malingerers may be correct, there will be others in which it will be wildly astray. There are illnesses which, in their early stages, present a convincing picture of malingering. An intimidated or over-conscientious worker might well be discouraged by overmuch propaganda from seeking treatment in time. There are inherent dangers in this approach and it may lead to suspicion and recrimination and, eventually, to much worse morale. Some malingering is to be expected, but it seems unlikely that conscious malingering contributes much to the total increase. We believe that it is not worth trying to tackle this problem from the point of view that it is to any large extent a reflection of malingering or deliberate abuse. We have little faith

that remedies based on exhortation or censure will have any permanent effect. If a scheme is unworkable without such measures there is something wrong with the scheme. It is doubtful whether the cost of more elaborate measures against malingering would be repaid even if all malingering were suppressed.

It is outside the scope of this investigation to try to judge whether the generosity of the scheme as it stands is justified by long-term considerations. It may be that increased production and efficiency and better general health will, in the long run, offset the cost of unnecessary absence and justify the continuance of the scheme even as it stands. But it should be possible to devise some modifications which, while preserving the principle of the scheme, would abolish some of the anomalies. A revision of the scheme was in fact suggested involving a good attendance bonus, offset by a small contribution from the worker towards sick pay. It was hoped that this might provide a monetary incentive to refrain from short absences, and it was coupled with a scheme for stricter supervision of longer absences. This particular suggestion was, however, found unworkable by departments.

Some further suggestions were discussed in detail and are summarized here.

The National Insurance certificate, which is used for the sick pay scheme, could be altered to allow the doctor to state that the patient was unfit for the kind of work he had described himself as doing. This would cover one possible cause of unnecessary absence, where a patient, probably in good faith, may misrepresent the nature of his work; for example, he may exaggerate its strenuousness or exposed conditions. We came across instances where this had happened, and such an alteration would allow some check by the employer and might help in finding alternative employment.

We suggested that the certificate could be altered to allow the doctor to state the number of days he considered the man would be off work and that the final certificate of fitness to resume work could often be omitted. Such an alteration would be likely to reduce the length of absences and also reduce attendance at crowded surgeries. We have learned that this alteration was already being considered and has now been introduced.

In view of the dangers of shop stewards' committees or other lay bodies carrying out checks on unnecessary absence, we discussed the possibility of making the payment of benefits for all absences over a certain length automatically dependent on the agreement of the factory medical officer, who would be the authority for continued payment. While there are difficulties in introducing such a change, when the present scheme is so closely interwoven with the administration of the National Health Service, our estimates suggested that the introduction of the change would not be difficult or costly for the factories themselves. The suggestion really introduced a change in principle by which it would be recognized that the employer should have some say in the disposal of his funds and the patient slightly more obligation to support his claim on the employer. This transference of authority for payment for the longer absences would clear up difficulties of medical etiquette, and the private doctor would be relieved of some of the conflict which faces him when he is asked to accept responsibility towards industry as well as to his patient.

It is a matter of great difficulty to try to assess the effect of any changes in a scheme of this kind on the basis of comparisons made before and after the introduction of the change. It is necessary to wait at least two years to begin to be sure of what is really happening as a result of the change. Other changes, such as those which might be made in the administration of National Health Insurance, or the occurrence of epidemics, would make it necessary to wait even longer.

We strongly recommend that changes should be introduced experimentally by trying them only on some of the establishments and keeping others, performing similar work in similar conditions, as experimental controls.

Problems arising from the introduction of sick pay schemes or of any forms of health insurance are of great importance to industry as a whole, but the investigation of these problems is of still wider interest in as much as they relate to other matters, such as the administration of National Health Insurance. With so large a number of Government establishments, all under very similar systems of administration, there is ample opportunity to investigate these problems more thoroughly, and these opportunities will be largely wasted by the wholesale introduction of one change or another.

Summary

A scheme for paid sick leave was introduced for Government industrial employees in September, 1948.

Records of sickness absence and other relevant information were collected from samples of the population in 31 Government establishments. Analysis showed the increase in sickness absence in the two years after the scheme was introduced to have been greater in men employed on time rates than in

those receiving some form of incentive payment.

A broad grouping of occupations into skilled or unskilled showed no real difference.

Age was associated with high absence rates, the registered disabled showed higher absence rates, and there was a disproportionate amount of absence taken by those about to leave. In none of these categories were the numbers sufficiently large to make their contribution to the total increase worth special attention.

The present sick absence rates for industrial workers on incentive payment are similar to those of non-industrial workers whose sick pay scheme is of long standing.

Observations made during visits to establishments, and the comments and suggestions of both workers' representatives and of management were taken into account in the conclusions.

This work was carried out under the general direction of Professor Sir Frederic Bartlett, C.B.E., F.R.S.

We are most grateful for the valuable assistance and criticism of Mr. J. W. Whitfield, lecturer in psychology, University College, London.

We have received the utmost cooperation from workers and management in the establishments we visited. We are especially grateful to the officers responsible for the arduous and monotonous task of producing our records and for the care with which this was done. We greatly appreciate the help given by the Ministries concerned.

Finally, we warmly acknowledge the assistance we have received from the Treasury, and would like to thank the two officers who assisted us in examining the records.

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THE ROLE OF STAPHYLOCOCCAL INFECTION IN BEAT DISORDERS OF MINERS

BY

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Since the report of Collis and Llewellyn (1924) the incidence of beat knee and elbow per 1,000 underground workers has increased more than tenfold. It is evident that the ineffectiveness of preventive measures which they noted has not been overcome. In 1949, 9,900 cases of beat knee and 1,500 of beat elbow were certified. Some of the increase since 1922 may be due to slight changes in defining the disorders and greater readiness to seek attention. However, a substantial part appears real and attributable to both the more frequent working of narrow seams and the increased use of mining methods which involve more kneeling. Despite their importance as the second most common of all the prescribed industrial diseases, little work on these disorders has been published. The problem has recently been discussed by Watkins (1951), who emphasized the importance of early treatment in view of the failure of current preventive measures.

In contrast to the more general survey of Collis and Llewellyn the present work attempts to identify the factors responsible for beat conditions by a detailed study, lasting a year, of men employed in a single low seam where a high incidence of the disorders was expected. Much of the investigation was bacteriological, for previously the traumatic element in pathogenesis had received more attention, to the especial neglect of the study of individual predisposition. The importance of this predisposition will be shown below and evidence submitted that it depends upon susceptibility to staphylococcal infection, particularly of hair follicles.

Working Environment

A newly opened seam of thin section, which was to be manned by workers unaccustomed to such conditions, was selected for this study.

The few men who had ever worked in low seams had not done so in the previous 12 months. There were approximately 215 men employed for more than a month at the coal face of whom 75% volunteered for observation. Most of the men were observed from the time production began. The district chosen had two double-unit conveyor faces in a seam varying from 2 ft. to 4 ft. in thickness except for occasional stretches with more height. Continual kneeling at work was necessary throughout the period of observation, except that in one section standing was possible during the third quarter of the survey.

In all faces the seam was constantly intersected by a band of " brass " (iron pyrites) but the coal itself was comparatively clean. Ash content averaged 26%, and volatile 21%. Although microscopically no excess of fusein was evident, mineral particles other than coal were conspicuous. The coal fragmented on handling into very hard pieces with noticeably sharp angles. This textural quality of the coal, together with the admixture of " brass ", meant that the men had to kneel on highly abrasive material. The floor itself was of moderately hard fireclay with soft patches, and had an irregular surface. In the first half of the survey water was constantly on the floor in one of the four faces.

Ventilation rates were 2,300–4,000 cu. ft./min. during the first eight months. Corresponding values for air velocities and temperatures are not available, but working conditions appeared comfortable. In the latter four months an air bridge permitted ventilation rates of 6,000 to 12,000 cu.ft./min., and temperatures were 68°F. dry bulb and 64–65°F. wet bulb (relative humidity 80–85%).

It was difficult to observe the men since they could be seen only outside working hours. There was no pithead bath at which the men could be seen with clean skins, but fortunately they cooperated well.

Special arrangements were made for the initial interview, many men being seen in their homes after bathing to permit of an assessment of their skin

condition. At this interview histories were taken of previous staphylococcal infection, beat disorders, and of previous working conditions. The men were assigned, according to their type of skin, to one of the four groups described below. Swabs were taken from nose, throat, and skin, and any abnormality of knees or elbows was noted. Skin swabs were taken from the front of the knees.

During the subsequent 12 months each man was examined as far as possible at monthly intervals, and questioned about the condition of his knees or elbows, the use of special protective measures, such as knee pads or spirit applications, and alterations in working conditions. Swabs were taken from carrier sites (nose, throat, and skin). Any beat lesion, cut or septic skin lesion found on examination was swabbed, some beat lesions also being needled for culture. Special visits were made to men with "beats" who were absent from work and repeated when disability was prolonged. The survey began in June, 1950. In the following October and November, by which time almost all the workers had been recruited for the new district, the serum levels of staphylococcal α-antitoxin of the majority of the men were estimated. The attendance and accident records of face-workers were noted and the seam was visited in most months to note its height, condition of the floor, temperatures, ventilation, and other working conditions.

Although the survey was intended only for observation of the men at risk and intervention was avoided if possible, medical advice, especially for a developing lesion, could hardly have been refused if the confidence and cooperation of the men were to be retained. As the survey proceeded, men reported trouble earlier, and simple treatment, sometimes with temporary transfer to work off the knees, undoubtedly aborted or ameliorated many cases of beat disorder. More suitable employment was found for one of the men who was especially prone to beat disorders.

Bacteriological Methods

All swabs were inoculated directly on to 2·5% sheep blood agar. After 24 hours' incubation at 37°C. in air with 20% CO_2 haemolytic colonies of staphylococci were picked and examined for the production of coagulase and α-toxin. Only strains producing both factors were considered pathogenic. The techniques used and the criteria of pathogenicity adopted have been described and discussed fully elsewhere (Marks, 1952). Several beat lesions were not swabbed either because there was antiseptic ointment on the skin or because the patients could not be seen until the lesions had subsided. Nasal swabs were taken from one nares only. Skin swabs were moistened and rubbed over approximately 10 sq. cm. of the skin of one knee; the unaffected one was also tested when a beat knee was present. Carriage over the knee was thought to be more relevant to the investigation than at the other sites usually tested. On most occasions swabs had to be stored overnight at room temperature before culture. *Streptococcus pyogenes* was sometimes isolated from infected cuts but not from any beat condition in this survey.

The serum levels of staphylococcal α-antitoxin were estimated in 117 men. Titrations were conducted at the Lh/10 level with intervals of 20%, using rabbit red cells.

All strains of pathogenic staphylococci isolated during the survey were stored until its end.

Survey Findings

Clinical Types of Beat Disorder.—The Industrial Injuries Act recognizes two types of beat disorder, subcutaneous cellulitis and acute bursitis. The aetiology of beat hand, which is always a cellulitis, differs from that of beat knee and elbow and was not studied; only one case was encountered. Fifty-seven attacks of beat-knee and three of beat-elbow were observed during the survey: it does not appear necessary, however, to distinguish between these disorders. Of the 60 attacks, only 31, involving 25 men, were admitted for benefit under the Industrial Injuries Act. Twenty-four of the attacks for which benefit was awarded were considered to have arisen as a complication of follicular infection, one was acute bursitis and in six the aetiology was doubtful. The case of acute bursitis was the immediate consequence of a blow from falling stone. Every one of the 29 attacks of beat disorder for which benefit was not claimed was manifestly the sequel to an infected follicle. Many septic cuts and abrasions were observed but none initiated a beat condition. In no attack of the "folliculitis" type was there a history of special precipitating trauma. Case 13 is representative of the "folliculitis" group.

Case 13.—A fireman, aged 32, with a greasy skin and a history of recurrent boils, developed a beat knee. A heavy growth of pathogenic staphylococci was obtained from the lesion, which began as folliculitis over the left tibial spine, rapidly developing into cellulitis. After three weeks' absence, he worked for two months in a district where no kneeling was necessary. Soon after his return to the low seam he developed boils of the neck; swabs from nose, knees, and neck now gave heavy growths of pathogenic staphylococci. Two weeks later cellulitis developed round one of several infected follicles on the right knee. Nasal and lesion swabs were again positive. He lost another 12 days' work and then obtained a permanent transfer to another district where kneeling was not required. He was observed for six months more but he had no further trouble with his knees, although remaining a heavy nasal carrier.

The relationship between attacks presenting as acute bursitis and those with staphylococcal cellulitis is of some interest. Immediate extension to an underlying bursa was only seen in three attacks of cellulitis, but several men developed a bursal effusion a few weeks after the subsidence of a folliculitis or a cellulitic beat attack. When first seen, the six attacks of doubtful aetiology would have been diagnosed by most observers as acute bursitis. However, as the history of one of these attacks shows, continued observation in such instances may prove an infective origin.

Case 3.—A conveyor-turner, aged 40, with a very dry and hairy skin was observed to be a nasal and skin carrier for four months before he developed a tense, tender swelling over the left tibial spine. There was no preceding trauma or visible local skin infection but he had some mildly-infected hyperkeratotic follicles about the face. The condition was then considered to be an acute bursitis. However, when the swelling subsided, a small tender area of skin was detected, which appeared to be an infected follicle buried in hyperkeratinized skin. He returned to work after 14 days' absence, but five months later an obvious follicular infection of the same knee developed into a severe beat condition which yielded staphylococci of the same phage-type as had been consistently carried in the nose for 10 months. It is suspected from the course of this man's first attack and the subsequent history that the original diagnosis of acute bursitis was mistaken.

It seems probable that the role of follicular infection in beat disorder is often overlooked, trauma alone or other types of infection being mistakenly held responsible for acutely swollen and painful bursae. It is noteworthy that all the men suffering attacks of doubtful aetiology were heavy carriers of pathogenic staphylococci; in addition, one had a history of recent staphylococcal infection and another a greasy skin. As will be shown below, these are factors especially associated with folliculitis and the cellulitic type of beat attack.

Morbidity.—In analysing our results, we have seldom distinguished between beat cases admitted to benefit after three days absence from work and those needing little or no time off. Although benefit cases are usually the more severe, there is no other clinical difference between the two groups and whether men lose time or not often depends on other factors.

The occupational groups most affected by beat conditions, with their complements of men, were colliers (124), cuttermen (12), prop-drawers (10), conveyor-turners (6), and officials (13). During the survey period 41 men in these groups suffered from beat disorders, 597 days being lost by benefit cases alone (28 attacks in 22 men). In the same time they lost 1,821 days through injury. Other occupational groups contributed only five beat cases, including three benefit patients who lost 47 days' work. Loss of output due to absences of less than four days and the lowered efficiency of the many men who continued to work despite beat disorder was no doubt considerable. In addition, experience of the disorder is one of the factors prompting men to leave the mining industry.

Effect of Hardening.—It is widely believed that after a period of the work on the knees susceptibility to beat disorder diminishes. Variation in many factors during the year's study prevents a clear-cut answer to this question. A high incidence of beat disorder was certainly observed during the first two months that the men worked in the low seam. This appeared to be due to especially susceptible men rapidly falling victims; if the 13 men suffering more than one attack during the survey are excluded, the incidence of beat disorder varied little with length of exposure.

In 25 men absent a month or more for reasons other than beat disorder, there was a lower incidence than average in the three months following their return to work. No case of beat disorder occurred in the six weeks following a two weeks' strike after the end of the survey. It appears, therefore, that the apparent lowering of risk with time is due to the elimination of men especially susceptible to staphylococcal infection, by transfer, invaliding, or the acquisition of immunity in the bacteriological sense. Periods of rest from kneeling, rather than increasing risk, appeared to be beneficial.

Occupation.—The highest incidence (25%) of benefit cases occurred amongst the 28 cuttermen, prop-drawers, and conveyor-turners. The numbers are small but it does appear that men in these occupations incur a special risk from having to move about the coalface on their knees; other factors predisposing to beat disorder were not especially frequent amongst them. Their length of working-week (mean 5·1 shifts) was very little greater than that of the 40 packers (4·8 shifts) who contributed only one benefit case, but were much less obliged to kneel at their work. There was a moderate incidence in the 124 colliers, 18 benefit cases occurring among them.

Knee-pads.—Collis and Llewellyn (1924) advised the use of knee-pads as an important defence against beat knee. Few of the miners we observed wore pads, although patches of canvas were often used to protect trousers against wear. Sixteen men

wore strapped knee-pads at one time or other but most abandoned them after a few weeks; only one wore pads throughout the year. Pads did not protect one youth from an attack of beat knee after only eight days' work, nor another who had adopted them after two attacks.

Results of Bacteriological Investigation

In the sections which follow all beat cases are considered together without regard to severity or benefit claims. The analyses deal only with men seen on four or more occasions at intervals of at least a month, but where length of observation was irrelevant a few more subjects were included. Few men were observed for less than six months, many for 12 months. Since approximately 25% of the men working in the district studied did not volunteer, an accurate comparison of beat cases and unaffected men is not possible. Most comparisons have therefore been made between beat cases and a set of matched controls, each closely corresponding to a beat case in age, occupation, number of shifts worked, and period under observation. It is known that no man who evaded observation lost time owing to beat disorder, and it is unlikely that many minor affections went unreported.

Carriage of Pathogenic Staphylococci Related to Morbidity.—In the following account the terms "carriage" and "positive swabs" imply the isolation of pathogenic staphylococci. Two thousand two hundred swabs were examined. The results for swabs taken from carrier sites and the skin over beat lesions (designated "beat lesion swabs") are given in Table 1. Only initial beat lesion swabs from the first attack experienced are recorded and other types of lesion, such as boils or infected cuts, are not considered here. Carriage of pathogenic staphylococci was more than twice as frequent, at each site tested, amongst men developing beat disorder as amongst matched controls. The higher incidence in affected men was not secondary to their beat lesions, carriage being as frequent before lesions developed as after; nor was it due to disproportionate swabbing of heavy carriers. Pathogenic staphylococci were isolated from 59% of the initial beat lesion swabs, an isolation rate three times that for skin swabs from the same group of men and 11 times that for skin swabs from matched controls. More positive beat lesions swabs might have been obtained if certain of the men could have been seen before their lesions had subsided. In 30 (27%) of the 112 men swabbed at four or more sessions (mean, 6·0 sessions), carriage of pathogenic staphylococci was not found on any occasion. Of these 30 men, only six developed a beat condition compared with 36 of the 82 men showing carriage at one time or another.

TABLE 1
INCIDENCE OF PATHOGENIC STAPHYLOCOCCI IN RELATION TO BEAT DISORDERS

Source	Beat Cases			Matched Controls			All Unaffected Men†		
	Swabs Examined	No. Positive	% Positive	Swabs Examined	No. Positive	% Positive	Swabs Examined	No. Positive	% Positive
Nose ..	241	110	46	235	46	20	487	123	25
Throat	217	14	6	194	2	1	440	12	3
Skin ..	241	42	17	229	11	5	487	38	8
Beat* lesion	37	22	59	—	—	—	—	—	—

* Nine unswabbed beat lesions do not appear in the table.
† Includes matched controls.

The association of nasal carriage with beat disorder contrasts with its apparent lack of significance in staphylococcal infections of wounds (Williams and Miles, 1949) although skin carriage is associated with both conditions.

To determine whether men prone to beat conditions can be identified by their carriage rate, the results of the first two swabbings have been analysed (Table 2). Men found to be carriers on both occasions will be termed "heavy carriers". There were 24 heavy carriers amongst 45 beat cases (53%) but only five amongst the matched controls (11%). The interval between swabbings was one month or more.

Skin Type, Morbidity, and Carriage of Pathogenic Staphylococci.—At their first interview the men were assigned, according to their skin types, to one of four broad groups. Greasy and abnormally hairy skins are two which require no special explanation. Men with fair, transparent skins were allotted to a type designated "fair". The remaining type was termed "seborrhoeic"

TABLE 2
CARRIAGE OF PATHOGENIC STAPHYLOCOCCI AND SUSCEPTIBILITY TO BEAT DISEASE

Type of Subject	No. of Men	Subjects with Carriage* at					
		First Swabbing	%	Second Swabbing	%	Both First and Second Swabbings	%
Beat cases	45	28	62	31	69	24	53
Matched controls	45	12	27	10	22	5	11
All unaffected men	85	27	32	29	34	15	18

* Nose, throat, or skin, or any combination of these.

but included many men with but a slight tendency to seborrhoea. The incidence of beat disorder and heavy carriage in men of different skin types is shown in Table 3. Men with greasy skins were the most susceptible to beat conditions, 15 out of 26 being affected. There was little difference between the other groups, which altogether had 31 with beat conditions out of a total of 107. Heavy carriage was found in approximately one third of the men with greasy, seborrhoeic or hairy skins, but in only one of the 20 men with "fair" skins.

TABLE 3
RELATION OF SKIN TYPE TO SUSCEPTIBILITY TO BEAT DISORDER AND CARRIAGE OF PATHOGENIC STAPHYLOCOCCI

Group of Men	No. of Men	Type of Skin			
		Greasy	Fair	Hairy	Seborrhoeic
Beat cases.. ..	46	15	6	3	22
Matched controls..	46	6	7	4	29
All men	133	26	20	9	78
Heavy carriers ..	39	10	1	3	25

Serum Levels of Staphylococcal α-Antitoxin.—The serum α-antitoxin levels of 117 men were estimated, mostly after one or two months' clinical observation. The relationship of the findings to other factors studied is complex but may be summarized as follows. Men with high α-antitoxin levels (1·0 unit or more per ml.) had a very high incidence of beat disorders and staphylococcal infection in the period of the survey and the two years preceding it and carried pathogenic staphylococci more frequently than the remainder; most were aged 30 or less. It was concluded that high antitoxin levels were due to recent staphylococcal infections, indicating a previous lack of resistance. There was some evidence that men with high antitoxin levels following recent infection continued to be especially susceptible to staphylococcal infections.

Beat disorder in men with low and medium serum α-antitoxin levels (< 1·0 units per ml.) was often associated with heavy carriage of pathogenic staphylococci. In contrast this association was not evident in men with high antitoxin levels. The significance of these observations is uncertain, but they may indicate that the effect of carriage in men with high antitoxin levels is overshadowed by other factors predisposing to infection.

Staphylococcal Phage-typing.—In 15 men (10 beat cases and five with boils or septic injuries) pathogenic staphylococci were first isolated from carrier sites and subsequently from lesions. In 11 of these 15 men (six beat and five other cases) the phage-type of the infecting strain and of that previously carried was identical (10 cases), or both were untypable (one case). In a further 19 men (eight beat and 11 other cases) carriage was absent or not sought before the lesion appeared but was present concurrently or subsequently. In 14 of these (seven beat and seven other cases) the infecting and carrier strains belonged to the same phage-type (nine cases), or were both untypable (five cases).

From these results it appears that beat disorder and staphylococcal skin infections are usually due to strains of staphylococci previously harboured. As noted by Williams and Miles (1949), a proportion of the discrepancies in phage-type may have resulted from sampling a flora containing more than one type. Some persistent carriers yielded staphylococci of the same phage-type throughout the 12-month survey period.

Previous History and Susceptibility to Beat Disorders

Men with histories of previous beat conditions or recent staphylococcal infection suffered a high incidence of beat disorder during the survey period. Recent infection was defined as that occurring in the two years preceding the survey; more remote staphylococcal infection was not of prognostic value. The only infections of note encountered were boils and severe widespread folliculitis. In Table 4 the proportion of beat cases with such histories is shown to be twice that observed in matched controls, or in unaffected men in general. During the survey period beat disorders affected 11 of 19 men who had recently suffered from staphylococcal infection and 13 of 24 who had contracted a beat condition at any time previously.

Incidence of Beat Disorders at Different Ages

The effect of age on the incidence of beat disorders was not great; it is most clearly seen in a single occupational group. Thus, 13 of 39 colliers aged 20 or less developed a beat condition, compared with 12 of 52 colliers over 20 years of age. Factors predisposing to beat disorders were not distributed evenly amongst different age-groups. In the younger men many of the beat cases had greasy skins or a history of recent staphylococcal infection. These factors were less common in older beat cases, which, however, often had a history of a previous beat condition. The association of a heavy carriage of pathogenic staphylococci with beat conditions was prominent at all ages.

TABLE 4
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Type of Subject	No. in Each Group	Recent Staphylococcal Infection	Previous Beat Affection (of any date)	Men with History of (1) (2) or Both	% Incidence of Group (3)
Beat cases ..	46	11	13	19	41
Matched controls..	46	5	5	9	20
All unaffected men	82	8	11	18	22
Totals ..	128	19	24	37	29

Summarized Analysis of Predisposing Factors

In Table 5 the incidence of chief predisposing factors is compared in beat cases and unaffected men. Thirty-nine beat cases, but only 19 of the matched controls, possessed one or more predisposing factors, a difference which is highly significant statistically ($\chi^2 = 16{\cdot}8$, $P < 0{\cdot}01$). The difference is also significant when men with two or more factors are considered; 17 such men were found amongst the beat cases but only two amongst the matched controls.

The effect of predisposing factors is most clearly seen in the men working in the high-risk occupations already defined, more of whom came under close observation. If men with a relatively short exposure to risk (less than 120 shifts during the survey) are excluded there remain 125 in these occupations, of whom 105 were studied. Amongst the latter, 32 of the 58 men with predisposing factors developed beat disorder compared with four of 47 men lacking them. At least one of the 26 men who escaped beat disorder despite predisposition was affected shortly after the end of the survey. Amongst those who worked less than 120 shifts there were five beat cases, four of which had predisposing factors, multiple in two cases.

The mean number of shifts worked in the district by beat cases before lesions appeared is of some interest when correlated with predisposing factors.

(1) Greasy skins .. .. .. 61 shifts (N = 13 S.E. = 16)
(2) Recent staphylococcal infection or beat history of any date .. 66 shifts (N = 17 S.E. = 10)
(3) Heavy carriage as sole predisposing factor .. .. .. 111 shifts (N = 10 S.E. = 21)
(4) No predisposing factor .. .. 130 shifts (N = 6 S.E. = 35)

Groups (1) and (2) are not mutually exclusive. Work records were not available for five men.

TABLE 5
INCIDENCE OF FACTORS PREDISPOSING TO BEAT DISORDERS

Type of Subject	No. in Each Group	Number of Subjects with					No. of Men with Two or More Factors
		Greasy Skins	Recent Staphylococcal Infection	History of Beat Disorder	Heavy Staphylococcal Carriage	Any of Factors (1) to (4)	
Beat cases	46	15	11	13	24	39	17
All unaffected men*	82 (36)	11 (6)	8 (5)	11 (5)	15 (5)	34 (19)	9 (2)
All men	128	26	19	24	38	73	26

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Discussion

Since most attacks of beat knee or elbow are demonstrably staphylococcal infections, it is apparent that factors other than trauma are also concerned in their pathogenesis. These factors and the problems they present are common to both beat disorders and staphylococcal skin infections. Most staphylococcal infections of a single follicle quickly heal if the follicle and the surrounding skin are protected against friction and immobilized. In contrast, an infected follicle exposed to pressure, friction, and movement heals slowly or may give rise to cellulitis or a boil, probably owing to the severance of the tissue barrier which normally can confine such an infection. It is widely believed that cuts and abrasions afford entrance to the organisms causing beat cellulitis, but although many such injuries were observed during the survey, none developed into a beat disorder.

In acute bursitis there is usually either a history of a severe injury to the bursa or an extension to it of infection from an adjacent cellulitis. The causes of chronic bursitis are less obvious. Collis and Llewellyn (1924) considered chronic bursitis to be unrelated to beat disorders. Since we have observed that effusions sometimes develop in knees recently the seat of follicular infections, further investigation of the aetiology of chronic bursitis seems necessary.

Preventive measures against beat disorders may be directed at either the infective or traumatic factors in their aetiology. In British coalmines the use of knee-pads to reduce trauma, although widespread, is failing to stop the steady increase in the incidence of beat knee. It is probable that pads increase liability to follicular infection because of skin maceration beneath them. Coal particles inevitably accumulate under the pads and reduce protection against trauma. Unless these deficiencies of knee-pads can be overcome, no substantial benefit from extending their use is to be anticipated.

In planning operations in a low seam the influence of duration of exposure to trauma, i.e., the time spent

but included many men with but a slight tendency to seborrhoea. The incidence of beat disorder and heavy carriage in men of different skin types is shown in Table 3. Men with greasy skins were the most susceptible to beat conditions, 15 out of 26 being affected. There was little difference between the other groups, which altogether had 31 with beat conditions out of a total of 107. Heavy carriage was found in approximately one third of the men with greasy, seborrhoeic or hairy skins, but in only one of the 20 men with "fair" skins.

TABLE 3
RELATION OF SKIN TYPE TO SUSCEPTIBILITY TO BEAT DISORDER AND CARRIAGE OF PATHOGENIC STAPHYLOCOCCI

Group of Men	No. of Men	Type of Skin			
		Greasy	Fair	Hairy	Seborrhoeic
Beat cases.. ..	46	15	6	3	22
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In planning operations in a low seam the influence of duration of exposure to trauma, i.e., the time spent

upon the knees, should not be overlooked, especially in occupations with the most risk.

The application of hardening agents to the skin is of doubtful benefit. Control of the infective element in beat disorders appears to offer most hope of reducing the incidence.

Little is known of the factors which determine susceptibility to staphylococcal infection. Such infection is most frequent in the post-pubertal period, as seen in the younger miners of this series, suggesting a hormonal effect on resistance. Ointments containing hormones have been advocated for the prevention of skin infection (Whitelaw, 1951) but there is insufficient evidence to justify their use in miners. Most experience of staphylococcal infection is eventually followed by an increase in resistance to further infection but the mechanism by which immunity is gained is obscure. Whether immunization with staphylococcal vaccines or toxoid would help to protect men exposed to risk of beat disorder can only be determined by a controlled trial. Reduction in the carriage of pathogenic staphylococci, even solely in the injured areas, is likely to be difficult, since those staphylococci harboured in the follicles are inaccessible to any reasonable form of cleansing or antibacterial treatment. Indeed, the vigorous scrubbing often advised for preventing skin infection may cause harm by injuring infected follicles. Nevertheless, investigation into the effects of regular application of alcohol and other antiseptics would be desirable.

The present investigation has indicated that men differ widely in susceptibility to beat disorders, and that predisposition can be estimated with some success by simple methods.

It may not be possible, however, systematically to exclude susceptible individuals from work in low seams, since alternative work in standing height is not often available. Nevertheless, it should now be possible to recognize those persons for whom preventive measures are especially necessary.

Supervision of men at risk, and especially of those adjudged susceptible, in order to detect and treat infection while it is still confined to the neighbourhood of the follicle, offers perhaps the most practical approach to the problem. An important therapeutic measure in most cases is temporary transfer to work off the knees.

The carrier rates for pathogenic staphylococci found in this survey were slightly lower than those recorded by other workers. This is partly due to our stricter criteria of pathogenicity and to avoidance of enrichment methods of culture which, by revealing very scanty staphylococci, might have masked an association of heavier carriage with beat disorders. Higher rates of skin carriage might have been obtained if sites other than the knee had been tested but they would have been less relevant to this investigation.

Summary

Beat disorders and the factors causing them have been studied for a year in men working in conditions particularly hazardous in this respect.

Fifty-three attacks of beat knee and beat elbow arose from the extension of an obvious staphylococcal infection of a hair follicle into the adjacent subcutaneous tissue. There were seven attacks of apparently primary (acute) bursitis, one clearly the result of a severe injury. In some of the remaining six, the bursitis had been preceded by a local follicular infection.

In no attack of beat disorder observed in this series did a cut or abrasion appear to afford entry to the infecting organism.

Most beat conditions occurred in men who could be recognized as especially prone to staphylococcal infection. Of the 46 men who developed beat disorders while under observation, 39 had recently suffered from a staphylococcal infection, had previously had beat disorders, possessed a greasy skin, or were heavy carriers of pathogenic staphylococci. The remaining seven men suffered only mild attacks.

The complete exclusion of such susceptible men from work in narrow seams may seldom be practicable, but their recognition should assist the application of other preventive measures.

Phage-typing has shown that the strains of staphylococci responsible for infection are usually the same as those previously harboured at carrier sites.

We owe much to the cooperation of local officials of the National Coal Board and of the National Union of Mineworkers Lodge. Mr. T. Thomas, pit first-aid attendant, gave especially valuable help. Mr. G. Harries and Mr. C. H. H. Thomas ably assisted in the clerical and technical work.

We are indebted for helpful criticism of this paper to Dr. Scott Thomson and to Dr. R. E. O. Williams who also undertook the phage-typing.

Dr. E. Lewis Faning kindly helped in the presentation of the statistical data.

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STUDIES ON COTTON DUST IN RELATION TO BYSSINOSIS

PART III: COMPARISON OF COTTON DUST AND HOUSE DUST BY CHEMICAL AND SKIN TESTS

BY

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In an earlier paper Cayton, Furness, and Maitland (1952) showed that extracts of cotton dust produced two types of skin reaction, an early reaction of wheal and flare and a late reaction of induration and erythema. These two reactions appeared to be caused by different substances. The late reaction was possibly a direct effect of some toxic component. The early reaction was of the type associated with allergy of the asthma-urticaria type, but it did not denote specific hypersensitivity peculiar to byssinosis because other groups of the adult urban population reacted equally with byssinotics although they had never been exposed to cotton dust and did not suffer from any allergic symptoms. The early reaction may, however, have indicated hypersensitivity to an allergen that was widely distributed.

In continuing this work preliminary chemical testing of cotton dust extracts suggested the possibility that they might contain similar components to those reported for house dust extracts by Rimington, Stillwell, and Maunsell (1947). House dust extracts also elicited the early type of skin reaction (Maunsell, Whetnall, and Rimington 1947). The further comparison of a cotton dust extract with house dust extracts is the subject of this paper.

Chemical Tests

Examination of Cotton Dust Extract.—Three extracts obtained by different methods from one sample of cotton dust were examined qualitatively by a number of chemical tests (Table 1). The details of preparing the extracts (designated I, II, and III) and the source of the dust will be found in an earlier paper (Cayton and others, 1952). The methods of extraction were respectively, that of Prausnitz (1936), a modification of this, and the method of Cooke (1947). These extracts behaved alike. Only the Molisch test and precipitation with phosphotungstic acid were positive. The biuret test was negative even when applied to extract II after it had been concentrated about seventeenfold. The results would be consistent with the presence of a carbohydrate possibly associated with a protein moiety.

TABLE 1
RESULTS OF CHEMICAL TESTS APPLIED TO EXTRACTS

Test	Cotton Dust		House Dust
	Extracts I, II, III, and X	Purified Extract X	Standard Purified Antigen
Molisch	+	+	+
Biuret	−	−	−
10% Phosphotungstic acid, precipitate	+	Sl.	Sl.
Tannic acid	−	−	Sl.
Trichloracetic acid	−	−	−
Sulphosalicylic acid	−	−	−
Glacial acetic acid	O	−	−
Ninhydrin	−	−	±
Orcin	−	−	−
Phloroglucinol	−	−	−
Methyl pentose	O	−	−
$BaCl_2$ (after hydrolysis)	O	−	−

O = not tested. Sl. = slight. ± = faint positive.

The corresponding extracts of house dust, the "standard crude antigen" of Rimington and others (1947), had given a positive Molisch reaction and a negative or doubtful biuret reaction, so that the two kinds of dust, extracted by different methods, had yielded extracts that were, in these respects, similar.

Extract X, the details of which were stated by Cayton and others (1952), was examined more fully. It was made by a modification of Cooke's (1947) method. After the dust had been de-fatted it was extracted with Coca's (1922) buffered saline solution, partially clarified in the centrifuge, and dialysed against Evans' (1922) saline for four days, changing the saline daily. The extract was then finally clarified in the centrifuge and sterilized by filtration through a gradacol membrane.

Nitrogen was determined by the micro-Kjeldahl

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method. Combined hexose was determined by the orcinol method (Rimington, 1940) using galactose as the standard. Reducing sugars were determined by the method of Somogyi (1945). Ashing was carried out at 500°C. in platinum crucibles and the ash weighed as sulphate.

This extract, which was soluble in water, conformed with extracts I, II, and III (Table 1) in giving a strong Molisch reaction, and negative biuret and ninhydrin reactions. It afforded the following analytical figures which are shown alongside those for "standard crude antigen" reported by Rimington and others (1947).

	Extract X	Standard Crude Antigen
Hexose (as galactose)	20·5%	20–30%
Nitrogen	2·5%	2–3%
Ash (as sulphate)	45·0%	35–50%

Examination of Purified Extract.—Another sample of extract X, prepared as described above, except that in this instance it was dialysed against running water instead of Evans' saline, was purified by the same method as Rimington used for the standard crude antigen. The finished extract was dialysed further against 2% (w/v) citric acid for 48 hours. This was followed by acetone precipitation. Acetone to 25% concentration was added and the solution stood overnight at 4°C.; no precipitate was obtained. The acetone concentration was increased and a small amount of precipitate was obtained at 50% concentration. This was discarded and the precipitate obtained at 75% concentration was centrifuged, washed with acetone, and dried *in vacuo*. This was the purified extract. No further precipitate was obtained at an acetone concentration up to 90%.

The material was yellowish brown and sparingly soluble in water. The results of colour and precipitation tests agreed in the main with those reported for "standard purified antigen", the difference being that the house dust extract gave a faintly positive ninhydrin test for free amino-groups and a slight precipitate with tannic acid (Table 1). The analytical figures obtained for the purified extract are shown alongside those for "standard purified antigen".

	Purified Extract X	Standard Purified Antigen
Hexose (as galactose)	49·2%	40–60%
Nitrogen	5·2%	5–7%
Reducing power (as galactose)	1·2%	1·5%
Ash (as sulphate)	1·0%	1–2%

Chromatographic Examination.—Purified extract X, in 10 mg. portions, was hydrolysed at 100°C. for 24 hours in 6N HCl, in sealed tubes, and the hydrolysate examined by the technique of partition chromatography (Consden, Gordon, and Martin, 1944). Phenol and butanol—acetic acid—water (40 : 10 : 50) were used as solvents. The amino-acids detected were cysteic acid (from cystine), glutamic acid, aspartic acid, serine, glycine, threonine, alanine, proline, valine, and leucine.

With the exception of cysteic acid these were the amino-acids that Rimington and his colleagues found in their "standard purified antigen" which contained in addition a little arginine and a trace of glucosamine. They were also the amino-acids present in the hydrolysate of materials, predominantly polysaccharide in character, isolated from moulds (Stillwell, Rimington, and Maunsell, 1947) except that proline was not found in the moulds.

To identify sugars, 10 mg. portions of purified extract X were hydrolysed for four hours at 100°C. with 0·5N H_2SO_4 in sealed tubes (Flood, Hirst, and Jones, 1947) and the hydrolysate examined by the procedure of Partridge (1948) using both phenol and butanol—acetic acid—water (40 : 10 : 50) as solvents. The papers were sprayed with the aniline-phthalate reagent of Partridge (1949), the naphtho-resorcinol reagent of Forsythe (1948), and the Elson and Morgan reagent for hexosamines of Partridge (1948). The main sugars identified were galactose, glucose, and arabinose. L-fucose was also present and traces of two sugars with RF values corresponding to ribose and rhamnose were detected. An unknown reducing substance, moving slowly in both solvents, was also found (Table 2). A yellow pigment remained stationary at the starting point in both solvents.

TABLE 2

^{R}F VALUES OF PURE SUGARS AND REDUCING SUBSTANCES PRESENT IN ACID HYDROLYSATE OF PURIFIED EXTRACT X

	Pure Sugars		Hydrolysate	
	Phenol	Butanol-Acetic Acid	Phenol	Butanol-Acetic Acid
Glucose	0·39	0·18	0·39	0·18
Galactose	0·43	0·17	0·43	0·17
Arabinose	0·51	0·25	0·52	0·25
Mannose	0·45	0·20	—	—
L-fucose	0·63	0·27	0·61	0·27
Ribose*	0·59	0·31	—	0·32
Rhamnose*	0·59	0·37	—	0·38
Unknown	—	—	0·08	0·07

* Figures extracted from Partridge (1948)

The report of Rimington and others (1947) that a similar analysis of "standard purified antigen" yielded only galactose has been amplified by more recent analysis (Rimington and Cummings, personal communication). Using finer methods, arabinose and xylose have also been found, together with small quantities of rhamnose and possibly traces of galacturonic acid and an unknown sugar, although some of the sugars present in small quantities might be impurities. The amino-acids were substantially as reported before and in the following percentages:—aspartic acid 6, glutamic acid 15·5, glycine 22, serine 16, valine 5, leucine 7, arginine or lysine 5, proline ±.

Thus the purified extract of cotton dust was similar, in its general properties, to the "standard purified antigen" obtained from house dust and to material extracted from moulds. All contained a polysaccharide associated with a polypeptide grouping of amino-acids giving none of the usual colour reactions for protein.

Skin Tests

In view of the chemical similarity of the cotton dust and house dust extracts, the skin reactions produced by them in the same persons were compared.

The technique adopted for the intradermal test was described in an earlier paper (Cayton and others, 1952) and was used throughout unless otherwise stated. The amount injected was 0·02–0·03 ml. and saline was always injected as a control. The dilutions referred to were made from a 1/1000 w/v solution unless otherwise stated.

Heat Stability of Cotton Dust Extracts.—The cotton dust extracts used for skin testing as reported in the paper mentioned above had been sterilized by filtration, with the exception of extract IV, which was prepared after the method of Rimington and others for house dust and immersed in boiling water for 30 minutes on each of three successive days. It appeared to be as active as filtered extracts in causing skin reactions. To confirm this, and to test the heat stability of the active substance at a temperature which would kill bacterial spores present in cotton dust (Furness and Maitland, 1952), samples of a 1/100 dilution of extract X, unheated and autoclaved at 10 lb. for 20 minutes, were compared by injecting them intradermally into each of a number of normal persons. These samples were alike as regards the incidence of early reactions they produced, and the size of reactions was also similar, as shown below. The heat stability of cotton dust extract was therefore another point of resemblance with house dust extracts.

Unheated Extract		Autoclaved Extract		Saline Control	
* 6	32†	7	28	—	
7	7	7	7	—	
6	19	5	5	—	
10	35	8	35	3	17
9	35	8	35	—	
5	23	4	15	—	

* Diameter of the wheal in mm. † Diameter of the whole reaction, including erythema, in mm.

Activity of Purified Extract X.—The activity of the purified extract X, 1/100 and 1/10, was compared with the original extract, 1/100, by testing them on 33 normal persons. The purified extract was adjusted so that the hexose content of the two extracts was similar on the assumption that the biologically active substances might be associated with the carbohydrate complex. The purified extract contained therefore about 2/5 as much total solid as the original extract.

The 1/100 dilution of purified extract caused fewer and smaller reactions of the early type than the same dilution of the original extract, but the 1/10 dilution of purified extract was practically identical with the 1/100 dilution of the original. Some typical examples of the comparative tests are shown below.

Although the purified extract was somewhat weaker than the original extract in causing the early type of reaction, it was still very effective.

As judged also by the late reaction, the purified extract was somewhat weaker than the original. Comparing a 1/100 dilution of each, the purified extract produced fewer and smaller reactions. The 1/10 dilution of the purified extract was again about equal in activity to the 1/100 dilution of the original. It is, however, of interest that the substances causing both the early and late reactions were not lost during purification.

Original Extract 1/100		Purified Extract 1/10		Purified Extract 1/100		Saline Control
*10	25†	9	35	7	32	—
6	18	6	23	—		—
6	14	7	18	0	8	—
11	30	9	28	8	22	—
5	17	5	17	—		tr.

* Diameter of wheal in mm. † Diameter of whole reaction, including erythema, in mm.

Comparison of Extracts of Cotton and House Dust by Skin Tests on the Same Persons.—Extract X (not purified) was tested by intradermal injection in comparison with a corresponding extract of house dust, the "standard crude antigen", also known by the trade name "domogen" which is a 1/1000 (w/v) solution of dried extract. This solution will be referred to as S.C.A. Extract X was calculated to be approximately the same strength. Dilutions mentioned subsequently were made from these solutions.

Both early and late skin reactions were recorded. The early reaction, read about 10 minutes after injection, was positive if there was a wheal (usually over 5 mm. across) and erythema with an overall diameter of 10 mm. or more. The late reaction was read about six to nine hours after injection and was positive if 5 mm. or more across. A control was considered to be positive if there was, for the early reaction, a wheal and flare of 10 mm. and, for the late reaction, a red thickened area of 4 mm. The appearances and the reading of these reactions were fully described in an earlier paper (Cayton and others, 1952).

Forty-three adults, including departmental staff, students, and some hospital patients, were tested. None had been exposed to cotton dust or was suffering from any allergic complaint. Two cases were excluded on account of their control reactions.

Early Reactions.—In analysing the results of the early reactions it will be noted (Table 3) that in the first seven cases extract X 1/10 was compared with S.C.A. 1/100. Five cases behaved alike to both extracts; two were positive, one reacted weakly (negative), and two had no reaction. Of the other two cases one was positive to each extract and negative to the other. There was not much difference in the size of the reactions in those who reacted to both.

In 34 cases (nos. 8–41) both extracts were used in a dilution of 1/10. The results were similar in 22 cases (15 positive, six no reaction, and one negative, weak reaction). There was a reaction with extract X only in 12 cases (nine positive and three small reactions classed as negative). When both extracts produced a reaction there was, on the whole, no significant difference in size or appearance. The results of the entire series can be summarized as follows:—

TABLE 3

COMPARISON OF EARLY AND LATE SKIN REACTIONS PRODUCED BY EXTRACTS OF COTTON DUST (EXTRACT X) AND OF HOUSE DUST (STANDARD CRUDE ANTIGEN) IN THE SAME PERSONS

Case No.	Early Reaction							Late Reaction		
	Extract X 1/10		S.C.A. 1/100				Saline Control	Extract X 1/10	S.C.A. 1/100	Saline Control
	Wheal (mm.)	Wheal + Erythema (mm.)	Result	Wheal (mm.)	Wheal + Erythema (mm.)	Result	Reaction (mm.)	Reaction (mm.)	Reaction (mm.)	Reaction (mm.)
1	7·5	22	+	7·5	18	+	–	9	–	–
2	7	33	+	4·5	16	+	–	13	–	–
3	tr(p)	–	–	5·5	19	+	–	12	–	–
4	3	7	–	3·5	8	–	–	14	–	–
5	–	–	–	–	–	–	–	15	–	–
6	–	–	–	–	–	–	–	3	–	–
7	4·5	10	+	4	–	–	–	6	–	–
		Extract X 1/10		S.C.A. 1/10				Extract X 1/10	S.C.A. 1/10	
8	5	25	+	7	36	+	–	15	–	–
9	8	34	+	7	25	+	–	tr.	–	–
10	8	22	+	7	28	+	–	12	–	–
11	9	41	+	10·5	43	+	–	tr.	–	–
12(1)	10·5	28	+	9·5	27	+	tr.	12	–	–
13(2)	9	30	+	13	40	+	6(p)	6	–	–
14	6	21	+	5	32	+	–	20	–	–
15	7	9	+	11	31(3)	+	–	20	–	–
16	7	30	+	6	22	+	–	–	–	–
17	6	15	+	10	26	+	–	5	–	–
18	6	20	+	5	15	+	4(p)	20	–	–
19	12	42	+	10	38	+	tr.	18	5	–
20	10	44	+	16	50	+	tr	22	–	–
21	7	31	+	6	28	+	–	tr	tr	–
22	7·5	26	+	7	20	+	tr	22	–	–
23	7	8·5	–	5	5·5	–	–	6	–	–
24	6·5	22	+	–	7	–	–	6	–	–
25	6·5	10	+	tr(p)	–	–	–	0	0	0
26	6	14	+	–	–	–	–	12	6	–
27	8	26	+	6(p)	–	–	–	tr	–	–
28	7	21	+	(p)	7	–	–	10	–	–
29	6	26	+	tr	–	–	–	45	–	–
30	8	41	+	–	10	–	–	8	–	–
31	5	16	+	–	–	–	–	13	tr	–
32	5	16	+	tr	–	–	tr	27	–	–
33(4)	5	8	–	tr	–	–	–	15	–	–
34	5	7	–	–	–	–	–	tr	–	–
35	4	10	–	–	–	–	–	5	–	–
36	–	–	–	–	–	–	–	10	–	–
37	4(p)	–	–	4(p)	–	–	3(p)	25	–	–
38	(p)	8	–	4(p)	–	–	4(p)	tr	–	–
39	tr	–	–	–	–	–	–	18	9	2
40	–	6	–	–	–	–	–	14	4	–
41	4(p)	17	–	6(p)	–	–	7(p)	23	–	–

(1) Suffered from asthma.
(2) Allergic to cat protein.
(3) Amount injected about 0·05 ml.
(4) Said to be sensitive to some drugs.

(p) = papule
tr = trace
0 = not recorded.

Reactions	Case Nos. in Table 3	Number of Cases
Both extracts positive	1, 2, 8–22	17
Both extracts negative (no reaction) ..	5, 6, 36–41	8
Both extracts negative (weak reactions)	4, 23	2
Extract X (only) positive	7, 24–32	10
Extract X (only) weak reaction (negative)	33–35	3
S.C.A. (only) positive	3	1

The two extracts gave the same result (positive, weak reaction or no reaction) in 27 cases (66%). Thirteen cases reacted to extract X only and one case reacted to S.C.A. only.

It is evident therefore that these two extracts, obtained by different methods from different kinds of dust, were similar; both caused an early type of skin reaction in normal adults, the reactions were indistinguishable, and, further, two-thirds of those tested gave the same result with each extract. The extracts were, however, not identical, for more people reacted to cotton dust than to house dust and, on the other hand, one person reacted to house dust only.

Late Reactions.—The two extracts differed markedly in producing late reactions, the figures being, for cotton dust extract 32 cases (80%) and for house dust extract four cases (10%). Three of the four reactions with house dust were small. The result with cotton dust agrees with earlier series of tests (Cayton and others, 1952). The result with house dust agrees with the opinion expressed by Maunsell (personal communication) that late reactions were probably rare, though the point had not been systematically investigated. It is clear however that the late reaction revealed a component in cotton dust extract which was absent or almost absent from the house dust extract.

Discussion

The comparisons of cotton dust and house dust extracts reported here have put the study of allergy to cotton dust in a new light. They have shown that it is not an isolated problem but is part of the wider study of allergy to other kinds of dust and in particular to house dust. This general conclusion arises both from the results of chemical examination and from skin testing.

Chemically, a similarity exists between cotton dust and house dust extracts in that both contain a polysaccharide associated with a polypeptide grouping of amino-acids giving none of the usual colour reactions for protein. Although the substances in these extracts have similar general properties they are probably not identical.

The dusts were different in origin and the methods of extraction were also different. The striking fact is that the extracts contained the same type of compound. There is evidence that the cotton dust extract was a mixture, even after its purification, although Rimington and others (1947) considered that the corresponding " standard purified antigen " from house dust contained specific antigenic material in a high state of purity.

Chromatography revealed that the two extracts had nearly the same amino-acids, and that they resembled in this respect material derived from moulds. The component sugars were similar but not identical in the two extracts.

The source of these polysaccharide-polypeptide compounds is not known but it could be moulds and possibly bacteria. The further chemical characterization and differentiation of substances causing the skin reactions, and the detection of their source is obviously desirable.

With regard to skin reactions the two extracts show a similarity and a difference. The difference is revealed by the late type of skin reaction which is produced by cotton dust extracts and not, or only slightly, by a house dust extract. The chemical nature of the substance causing this reaction is not known. It was thought to be an irritant or toxic substance acting directly on the tissues and not concerned in hypersensitivity (Cayton and others, 1952).

The similarity of the extracts is revealed by the early type of skin reaction. The two extracts tested gave almost identical results in 66% of individuals but the cotton dust extract caused a reaction in more persons than the house dust extract, and the house dust extract caused a reaction in one person negative to cotton dust.

It may be assumed that one or more polysaccharide-polypeptide compounds in the cotton dust extracts were responsible for the early reactions, as Rimington and others (1947) have shown that hydrolysis of these substances in house dust extracts resulted in loss of skin-reacting activity. It is of interest too that both extracts were active after being heated, the house dust at 100°C. and the cotton dust at about 115°C.

The comparative skin tests were made on normal adults who had not been exposed to cotton dust and did not suffer from allergic manifestations. It is known from our earlier observations that different groups of adults react similarly to cotton dust and therefore the comparison of the two extracts as here reported would be expected to apply to byssinotics, workers in cotton-mills not exposed to dust, and allergic patients who were sensitive to a variety of allergens.

Reactions to house dust of the early type are considered to indicate allergy, at least in some cases, and there is some supporting evidence for this view (Maunsell and others, 1947). In others, especially those who react only to stronger concentrations, such as we have used here, the significance of the reaction is in doubt. Stillwell and others (1947) wrote " at present we are unable to state whether a threshold reaction to dust antigen at this dilution represents a low grade sensitivity to dust (i.e. a weak reactor) or a non-specific reaction ". The dilution referred to was 1×10^{-4} w/v, equivalent to the 1/10 dilution of standard crude antigen used in this work. We have discussed the same problem regarding cotton dust extracts (Cayton and others, 1952) but were uncertain as to whether there was any qualitative difference between those who react to low dilutions and those who are much more reactive. A few persons react to much higher dilutions than others and they may be hypersensitive, but as the reactors to cotton dust may have no clinical manifestations of allergy it is necessary to obtain additional evidence from serology and laboratory tests before being certain how to interpret these reactions. Further work is in progress to determine which, if any, of the reactors (early type of reaction) are hypersensitive. It is clear that such researches on cotton dust have a general significance for the understanding of allergy associated with other kinds of dust and with moulds.

Summary

An extract of cotton dust has been compared chemically and by skin tests with extracts of house dust. Both contained a polysaccharide associated with a polypeptide grouping of amino-acids giving none of the usual colour reactions for proteins. Quantitative chemical analysis of crude and purified

extracts from the two kinds of dust showed a marked similarity. Chromatographic examination of the purified extracts, after hydrolysis, revealed a similarity in the sugars and amino-acids detected.

Both extracts produced the early type of skin reaction, and this property resisted heating at about 100°C.

Cotton dust extract produced a late skin reaction in a high percentage of those tested, whereas the house dust extract did not.

The two extracts were, therefore, not identical. Polysaccharide-polypeptide compounds were probably responsible for the early skin reaction. Their source is not known but they could come from moulds or possibly bacteria.

The significance of these results in relation to byssinosis and to allergy is discussed.

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AIRBORNE BACTERIA IN COTTON MILLS

I: SURVEY OF COUNTS OF VIABLE BACTERIA

BY

D. G. DRUMMOND and MARY HAMLIN

From the Shirley Institute, British Cotton Industry Research Association

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The pulmonary disorder known as byssinosis (Prausnitz, 1936; Caminita, Baum, Neal, and Schneiter, 1947) which affects the operatives in the card-rooms of cotton spinning mills is usually attributed to the high concentrations of dust in the card-room air. The concentration of airborne bacteria has, however, been found to be abnormally high in these rooms; and also the counts of viable bacteria and fungi in cotton waste extracted by machines and air-cleaning plant in mills spinning raw cotton (see also Furness and Maitland, 1952). Although byssinosis may take 20 years to develop and cannot be due to bacterial infection in the ordinary sense, the possibility of dead and viable microorganisms in cotton dust playing some part in its aetiology cannot be excluded.

The survey of counts was initially carried out in card-rooms in full production in eight spinning mills chosen to cover a variety of types: two mills spinning mainly Egyptian cotton, two mills spinning mainly American cotton, two mills spinning mainly Indian cotton, one cotton waste spinning mill, one rayon spinning mill. The results are given in Table 1, in which relevant data from later work have also been included. Results for other sites in the mills were also obtained, but those for spinning rooms and later stages of processing, including weaving, as well as those referring to rayon have been omitted from the table; they are sufficiently indicated in the outline of conclusions given below. Outside control counts were not usually made, but the cotton card-room counts are so high that such controls would in most cases have little significance.

A Bourdillon type slit sampler (Bourdillon, Lidwell, and Thomas, 1941) was used, and in the early work the plates were, generally speaking, very overcrowded so that systematic errors affected the counts per cubic foot. Corrections have been made, but random errors remain and are probably large, since the numbers of plates taken at any one site in our early work were small. This however does not invalidate the following broad conclusions.

In a normal card-room processing raw cotton on rotating flat cards the bacterial count among the cards in production is exceptionally high, running usually to several thousands per cubic foot.

In the same card-room, but among the speed frames and away from the cards, a much lower count of the order of 500 to 1,000 per cu. ft. is found. A similar reduction was found when the sampler was moved from cards processing cotton to others processing rayon staple fibre in the same room (Table 1, Mill No. 5). Even these lower counts are high by ordinary standards.

In the pre-carding processes on raw cotton (cotton mixing, blow-room) the counts are comparable with those between cotton cards in production in spite of the more enclosed nature of the machinery, but local variations of concentration are probably more marked than in the card-room.

In the spinning rooms the counts are mostly of the order of 50 to 200 per cu. ft., which are numerically comparable with those found in such rooms as a canteen or a crowded office, though as Hamlin shows below, the types of organisms present are still those characteristic of the card-room rather than of a room with a relatively high human population.

Plates taken in canteens and offices inside the mills show colonies which are obviously of different types from those found on plates from the cotton workrooms.

In a weaving shed the counts are of the same order as those in the spinning rooms.

In a mill working on viscose rayon staple fibre (with a little acetate rayon) the counts, even where the material was going through the rotating flat cards, were less than 10 per cu. ft., which is indistinguishable from an outside control count.

In the card-rooms of the cotton waste mills visited the maximum bacterial count was 230 per cu. ft. as compared with the thousands in an ordinary cotton card-room, and two factors may be respon-

TABLE 1
BACTERIAL COUNTS* IN COTTON MILLS

Mill	Type of Material	Bale Opening Room	Cotton Mixing Chamber	Devil-room	Blow-room	Card-room (Cards)	Card-room (Speed Frames)	Speed Frames (Separate Room)
	Raw Cotton							
No. 1	65% American (plus other cottons)	—	—	—	——	3,500	—	4,600‡
No. 1 (repeat visit)	58% American, 23% Egyptian (plus others)	—	—	—	1,200	1,800	—	580
No. 2	65% American (plus other cottons)	—	—	—	—	8,100	—	—
No. 3	25% American (plus other cottons)	—	—	—	—	6,500	—	—
No. 3 (same visit)	8 parts Indian plus 1 part Congo cotton	—	—	—	3,300	3,200	1,500	—
No. 4	100% Indian (Oomras)	—	—	—	12,000 / 2,600	2,700	—	—
No. 5	100% Egyptian	79	—	—	—	7,200†	290	—
No. 6	100% Egyptian	—	1,300	—	950	930	59	48
No. 9 (two visits)	100% Egyptian	—	—	—	—	2,700 / 3,400	—	—
Nos. 10–15 (mean of many visits)	American plus Brazilian	—	—	—	—	11,700	—	—
No. 16	100% Sudanese	—	—	—	—	4,800	—	—
	Cotton Waste							
No. 7	Mixed hard and soft	—	—	400	180 (scutcher)	41 (breaker cards) 31 (finisher cards)	—	—
No. 17	Mixed hard and soft waste	—	—	1,700	—	230 (breaker cards)	—	—

* Counts per cubic foot of air of organisms growing aerobically at 37°C on nutrient agar.
† Near cards on viscose rayon in the same room the count was 420.
‡ An abnormally high count, never again approached in such a room. The repeat visit gave a typical figure.

sible for this striking difference. In the first place the " hard waste " cotton has already been processed once and much of the trash and dust removed, and secondly these mills use roller-clearer cards instead of flat cards. (Roller-clearer cards are hooded and fitted with exhaust ducting so that much of the ejected material is carried off immediately and does not enter the general air of the card-room.) On the evidence available it is not possible to distinguish between the effects of these two factors, but the indications suggest that the exhaust system has the greater effect.

The counts at the pre-carding processes in a waste mill (devil-room, scutcher) are well above those in the card-room of the same mill and are high by normal standards, though still below those in ordinary cotton blow-rooms or card-rooms.

Discussion

It is legitimate to deduce from these results, taken in conjunction with those of the subsequent paper on the type distribution of the organisms, that the very high counts of bacteria in cotton card-rooms are associated with the dirt and trash which are imported with the cotton in the bale, and that the organisms originate in the soil of the cotton fields. Once the material has left the card-room the counts drop to a low level, though whether this is because the trash has been removed or because the cotton is in a less open condition is not certain; the higher count on reopening the spun cotton in the devil-room suggests that the latter factor is at least partly responsible. Cellulose which has been chemically dissolved and regenerated in the form of rayon fibres is virtually sterile.

Although the bacteria are associated with the dirt in the cotton as received, the ratio of the number of organisms to the number and mass of dust particles may vary widely with cottons of different origin or of different crop years. There seems, for instance, to be a definite tendency for American cottons to give a higher bacterial count and often a lower dust count than either Egyptian or Indian types (see Table 1, Mill No. 3, for a

comparison of counts between cards on American types and cards on Indian types in the same card-room), and we have recently obtained evidence of a marked increase in the bacterial content of certain American and Brazilian cottons in the 1950 season as compared with 1949.

Summary

Counts of airborne bacteria have been made at various sites in cotton mills. They are exceptionally high in the card-rooms and at pre-carding processes but much less at the spinning and later processes. They are comparatively small in a waste mill card-room but rather higher in the devil-room.

At all sites in a rayon mill the counts were indistinguishable from control counts.

In cotton mills the general level of counts depends on the source of the cotton and tends to be highest with American types; even for any one type it probably also depends on the crop year.

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II. DETERMINATION OF TYPES OF VIABLE BACTERIA

BY

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From the Shirley Institute, Manchester, British Cotton Industry Research Association

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An examination of the types of bacteria present in the air has been made in cotton mills using cottons of various origins. The organisms were collected on solid media in a Bourdillon type slit sampler and exposures were adjusted so that no plate from which subcultures were made carried more than 200 colonies. In all cases the medium was made up with 5% agar since at lower concentrations some colonies tended to spread.

Samples were taken in two cotton spinning mills. The first of these was visited twice and samples were collected in the card-room and at various other sites. Blood and nutrient agar were used in each case, and on the second occasion anaerobic as well as aerobic incubation was employed. One visit only was paid to the second mill and samples were only taken in the card-room; blood and nutrient agar plates were exposed, some being incubated aerobically and others anaerobically.

In order to find whether the distribution of genera changed substantially as the cotton reached later stages of processing, visits were also made to a waste spinning mill and to two weaving sheds. The results from the spinning mills had shown that there was no significant difference between the types found on blood agar and those on nutrient agar and also that no obligate anaerobes were detected; therefore on these later visits only aerobic cultures on nutrient agar were examined.

Details are given in Table 1.

After incubation colonies were subcultured from the aerobic plates to slopes, and from anaerobic plates to stabs; those from blood agar plates to blood and nutrient agar, those from nutrient agar plates to this medium only. It was found that all the organisms would grow well on nutrient agar slopes, and the cultures were therefore maintained in this way. The organisms were assigned to their genera according to Bergey's classification (Bergey, 1948). The results are given in Table 2. The numbers of colonies examined and on which the counts per cubic foot were based are shown in brackets. High absolute accuracy is not claimed and the figures are intended for comparison within the table. For the less abundant types of organisms the number of colonies was very small and here in particular the accuracy is low.

Discussion

The investigation showed that the bulk of the organisms in the card-room air belonged to those genera which are commonly associated with soil and water, especially the former. No true anaerobes were isolated, and organisms commonly found in the respiratory tract were virtually absent.

The genus *Bacillus* always predominated in the aerobic cultures, but the genus next in order of abundance has varied. The data are not sufficiently extensive to show whether the variation arose from differences in mill conditions or from the different mixings of cotton.

The total count based on anaerobic cultures was one-fifth or less of that obtained with aerobic cultures. *Bacillus* was very much suppressed but

TABLE 1

No. of Mill†	1 (1st Visit)	1 (2nd Visit)	16	17	18 (1st Visit)	18 (2nd Visit)
Work in Progress	Cotton spinning	Cotton spinning	Cotton spinning	Waste spinning	Weaving	Weaving
Sites at which Plates were Exposed	Card-room Blow-room Speed frame room	Card-room Ring-room	Card-room	Card-room Devil-room	Weaving shed "T"	Weaving shed "O"
Type of Cotton	58% American 28% Egyptian 14% other growths	100% American	100% Sudanese	Mixed waste	American	Mixed. Sampler sited among looms weaving Egyptian cotton
Culture* Conditions	N.A. } 37°C. B.A. } 48 hrs.	N.A. } 37°C. B.A. } 96 hrs. N.An } 37°C. B.An } 72 hrs.	N.A. } 37°C. B.A. } N.An } B.An } 72 hrs.	N.A. 37°C. 90 hrs.	N.A. 37°C. 90 hrs.	N.A. 37°C. 90 hrs.
Notes	Sampling poor owing to small number of plates examined	Number of plates increased to improve sampling	Number of plates again increased to improve sampling	—	—	—

* N = nutrient agar ; B = blood agar ; A = aerobic incubation ; An = anaerobic incubation.
† The numbering of the mills is the same as that used by Drummond and Hamlin in the first paper of this series.

TABLE 2

NUMBERS OF ORGANISMS (COUNTS PER CUBIC FOOT)

Mill	Site	Type of Incubation	Total Count (per cu. ft.)	*Bacillus*	*Achromobacter*	*Flavobacterium*	*Aerobacter*	*Corynebacterium*	*Micrococcus*	*Actinomyces*	*Pseudomonas*	*Sarcina*	*Escherichia*	*Streptococcus*	Failed to Subculture or Died before Identification
No. 1 1st Visit	Card-room	A	1,800 (200)	910 (102)	330 (37)	170 (19)	120 (14)	44 (5)	18 (2)	18 (2)	18 (2)	—	—	—	150 (17)
	Speed frames room	A	540 (59)	200 (22)	130 (14)	100 (11)	36 (4)	9 (1)	27 (3)	—	—	—	—	—	36 (4)
	Blow-room	A	1,200 (124)	550 (59)	230 (25)	190 (20)	65 (7)	—	93 (10)	—	—	—	—	—	28 (3)
No. 1 2nd Visit	Card-room	A	6,200 (235)	3,900 (149)	110 (4)	27 (1)	580 (22)	260 (10)	110 (4)	110 (4)	—	—	27 (1)	—	1,100 (40)
	Card-room	An	1,600 (202)	210 (27)	300 (38)	360 (46)	300 (38)	47 (6)	8 (1)	—	—	16 (2)	40 (5)	—	310 (39)
	Ring-room	A	220 (245)	140 (152)	5 (6)	1 (1)	15 (17)	12 (13)	6 (7)	—	—	1 (1)	—	—	43 (48)
	Ring-room	An	41 (200)	2 (8)	9 (43)	16 (80)	8 (37)	1 (5)	<1 (1)	—	—	—	—	—	5 (26)
No. 16	Card-room	A	2,900 (480)	2,300 (390)	120(21)	—	—	12 (2)	42 (7)	230 (38)	—	—	—	—	130 (22)
	Card-room	An	170 (136)	42 (34)	16 (13)	73 (59)	15 (12)	1 (1)	1 (1)	1 (1)	—	1 (1)	—	1 (1)	16 (13)
No. 17	Card-room	A	230 (400)	220 (376)	2 (4)	—	2 (4)	1 (1)	8 (13)	—	—	—	—	—	1 (2)
	Devil-room	A	1,700 (400)	1,200 (277)	83 (19)	4 (1)	26 (6)	148 (34)	161 (37)	70 (16)	—	30 (7)	9 (2)	—	—
No. 18	Weaving-shed T	A	246 (200)	213 (173)	2 (2)	1 (1)	5 (4)	6 (5)	14 (11)	4 (3)	—	1 (1)	—	—	—
	Weaving shed O	A	148 (205)	87 (121)	11 (15)	5 (7)	3 (4)	12 (17)	19 (26)	4 (6)	—	3 (4)	—	—	4 (5)

A = aerobic. An = anaerobic

Number of colonies on which each figure is based given in brackets.
The nutrient and blood agar counts are not statistically significantly different and have been combined in this table. The counts are given to the first two significant figures.

the count of *Flavobacterium* was significantly larger. The latter was also true in some cases of *Achromobacter* and *Aerobacter*. The distinction between *Achromobacter* and *Flavobacterium* is slight and not always certain.

There can be no doubt that the genus *Bacillus* is predominant in the card-room and that the remaining organisms consisted almost entirely of *Achromobacter*, *Flavobacterium*, *Aerobacter*, *Corynebacterium*, *Actinomyces*, and *Micrococcus*. This is true also of the distribution of genera in the weaving sheds and waste mill in which no types were found which were not already represented in the card-room.

Statistical analysis of the aerobic counts of *Bacillus* expressed as percentages of the total aerobic counts did not establish any significant difference between the two visits to Mill No. 1 (the data for the first visit are rather sparse), although the cotton mixing had been altered ; the increase in percentage on moving to Mill No. 16 was significant at the 1% level.

The airborne organisms in other parts of a cotton spinning mill were similar to and had much the same percentage distribution as those in the card-room of the same mill on the same occasion, though the total count per cubic foot might be different. This, however, was less true of the waste mill, where the devil-room showed a much higher count than the card-room, but a lower percentage of *Bacillus* which was statistically significant, though it remained much the most numerous type.

Summary

A study has been made of the relative incidence of the genera of bacteria present in the air of cotton mills. *Bacillus* is the most prominent, followed by *Flavobacterium*, *Achromobacter*, *Aerobacter*, *Corynebacterium*, *Actinomyces*, and *Micrococcus*. No true anaerobes have been isolated and organisms characteristic of the respiratory tract were rarely found.

This form of distribution was essentially the same at all stages of processing although the number of organisms per cubic foot was very different.

Reference

Bergey, D. H. (1948). " Manual of Determinative Bacteriology ", 6th ed. ed. Breed, R. S., Murray, E. G. D., and Hitchens, A. P. Baltimore.

BOOK REVIEWS

Annual Reports of the Chief Inspector of Factories for the Years 1949 and 1950. Cmd. 8155 (4*s.* 6*d.*) and Cmd. 8445 (8*s.* 6*d.*) London : H.M.S.O.

The Annual Report of H.M. Chief Inspector of Factories is a document that has considerable interest for a wide variety of people, and the two reports under review are no exception.

The industrial medical officer might be tempted to think that his interests are limited to that part of the report dealing with industrial disease—20 pages in the 1949 report and 24 in the 1950 report. While he would obviously be wrong to omit these pages he would be equally wrong to assume that the rest of the report was without interest to him. Indeed, from the point of view of practical everyday work many of the sections under the heading "industrial disease" will be of academic interest only, as he will be unlikely to meet some of the diseases in a normal lifetime spent as an industrial medical officer. Turning first to these pages, however, we find that the decline in lead poisoning between 1900 and 1950 is a striking proof of the effective work that has been done in preventive medicine in industry. In 1900, 1,058 cases of lead poisoning were notified ; in 1949, 53 cases, and in 1950, 57, of which 27 came from the shipbreaking industry. In the pottery industry the figures are 210, two, and one respectively ; coach and car painting, 70, 0, and 0 ; white and red lead works, 377, one and four. Although a doctor may be responsible for the health of workers for whom there is no exposure to this particular risk, it would be a mistake to think that these figures have no particular relevance to his own problems. The methods by which the lead risk has been brought so largely under control are multifarious ; by replacing with harmless materials those which are toxic, by engineering practices of dust control, by medical supervision, and by attention to personal hygiene, by good personnel and welfare supervision. Some or all of these methods may be applicable to a particular problem that confronts a particular medical officer, notwithstanding the fact that he is not dealing with lead. Chromium plating, for example, is a relatively common process. It is carried out in large and small works. The number of cases of chrome ulceration notified has increased in the last 20 years. In 1940 there were 57 cases ; in 1949, 80 ; in 1950, 100. Many of the preventive measures are of an engineering rather than a medical nature and co-operative effort by doctor, engineer, and management is essential if it is to be effective.

In the 1949 report there is brief mention of a four-year survey of industrial lung disease of iron and steel foundry workers. Silicosis was found to be a serious risk among sand and shot blasters, fettlers of steel castings, furnace bricklayers, and some iron moulders. The 1950 report devotes 14 pages to a survey of health and welfare conditions in iron foundries. Although the general trends indicate a growing interest in and importance attached to the provision of good washing facilities, it is suggested that in some areas there is still a great deal of room for improvement. It is not only management that is at fault, however, and a distressing example is quoted of the misuse and abuse by the workers of newly-installed washing accommodation.

There is no doubt that there is still plenty of scope for the doctors to exert their influence both on management and on the workers in respect of ordinary standards of hygiene. Ignorance is a great enemy of good health. By instruction, by information services, and by publicity the industrial doctor can do much to ensure that management and workers are on the side of good health and not against it.

It is sometimes difficult to remember accurately anything of the industrial and economic conditions as recently as 12 months ago. The first 30 pages of the 1949 report give an interesting and informative description of the major industrial developments during the year. Naturally enough the highlights are picked out, and these indicate the gradual but steady progress that scientific methods are making in relieving human effort and replacing it by mechanization. This technical advance has both its advantages and its dangers to health. Mechanization in stacking relieves a source of human strain. In the porcelain and pottery industries new processes have reduced hazards of dust. But often hazards resulting from new methods can only be avoided if very careful thought is given to some of the problems created by new methods or new machinery.

In considering the physical environment of factories, both reports devote space to the general conditions of lighting, colour, temperature and ventilation, washing and clothing accommodation, and the various other general conditions which affect everyone of our industrial population. It is too easy to be attracted to the more exciting aspects of factory hazards and forget that, for example, the standard of washing accommodation or the general and local illumination is going to affect every single worker in the factory. It is good, therefore, to learn that increased attention is being paid to both natural and artificial lighting. There seems to be some doubt in the mind of the Factory Department in just which direction the trends are going. For example, in the 1949 report we read :

> "It is satisfactory to note that in the building of new premises and the reconstruction of old ones, consideration is being given to obtaining the maximum amount of natural light. Not only is there a saving in the cost of artificial light, as even on sunless days large glazed areas give sufficient natural light for most purposes,

but it is recognized that the psychological effect for good is very great. Further, as windows are seldom installed without some sections that open, there is considerable improvement in the natural ventilation."

whereas, in 1950 :

"In most new factories natural lighting has received its proper attention and is good, but there are still cases where this subject seems to have been low on the list for consideration and the windows appear to have been little more than an afterthought. Architects should bear in mind that daylight is the best light in which to work and so design windows and roof lights that the maximum advantage is obtained by the manufacturer, both as regards production and the general conditions of work."

While it is perhaps unfair to place these two paragraphs side by side it serves as a warning to the complacent. It is never possible, either over the country as a whole or in regard to a particular factory, to say, "Since now we have the standards that we have been aiming at we can relax our vigilance and attend to other things." The price of good environmental standards is eternal vigilance and the vigilance of the medical officer in these directions is no less important than his vigilance in regard to toxic hazards.

Much can be done in the architect's office at the drawing board stage to prevent problems, but if the architect is not in very close touch with engineers, doctors, executives, and operatives he may create problems that should never have arisen. Even lighting engineers may produce problems of comfort or create frustration by the very measures they take to provide good lighting. If, for example, the cleaning of windows, lighting fittings, etc. are not considered at the design stage the problem of cleaning may be overlooked and produce a major difficulty or incur a substantial expense.

Colour schemes continue to develop, and both on aesthetic and on functional grounds have much to recommend them. Many examples are quoted in both reports to illustrate the growing interest in this direction.

During the period under review new legislation has been enacted which requires the provision of seats wherever this is practicable. Since 1948 much publicity has been given by the Factory Department to this requirement with the result that much general interest has been aroused. It is heartening to read that managements have been ready to cooperate with one another by the exchange of information. There has been a gradual recognition of the relationship between seating as it affects comfort and as it affects general efficiency. By giving careful thought to the problem of the best method of seating firms have come to look at jobs from the point of view of economy of movement ; and alterations in design of benches, layout, and height or angle of work have resulted in further improvements in efficiency as well as greater confort.

Here again the doctor can bring his influence to bear on problems that are universal and fundamental and which reinforce the belief that industrial efficiency may often be a by-product of industrial well-being.

It is understandable that much of the work of the Factory Department is concerned with accident prevention. We must not be surprised therefore that nearly half of each report is taken up with this subject. But we are not asked simply to read a series of accident reports. In each report a selected type of accident (eye injuries, accidents caused by cranes, etc.) is discussed as well as accidents confined to certain industries. Besides giving factual statements as to the number of injuries, classified according to cause given, there is also valuable advice on accident prevention. Here again the doctor cannot stand aside from the problem and regard it as outside his province. Knowledge of the incidence and cause of accidents is essential to their effective prevention.

Finally, it can be said with emphasis that both the text and the tables which make up these two volumes are essential to the doctor who believes that his horizons are wider than his surgery walls, and that he has a part to play not only in preventing sickness but also in fostering the physical, mental, and social well-being of the population. H. G. MAULE

An Introduction to Psychology. By Gardner Murphy. (Pp. xvii + 583. 30*s*.) New York : Harper Bros. 1951.

There is something ominous about the word "introduction" in the title of any textbook ; when the subject is psychology, the portent becomes even more alarming than usual. This "ominous" quality, curiously enough, can be related to either of two extreme meanings : "dangerous and misleading over-simplification" or "an expert's euphemism for very solid groundwork stuff". Gardner Murphy, one of the most erudite and distinguished of American psychologists, already the author (among other works) of a popular brief text and a scholarly tome on personality, has this time avoided the grim extremes and given us a genuine and readable introduction ot his subject. To quote him, "My desire has been to try my hand at the task . . . of combining a clear and consistent viewpoint with a genuine interest in all sorts of psychological facts." He adopts as his basis the conceptions "that every psychological act is the act of a whole person" and "that each activity of a person can best be introduced not by describing the abstract process (e.g., perception, learning, thinking), but by describing a person carrying out such an activity". In 30 short chapters he covers the traditional topics in this "personalistic" way, first grounding everything firmly in the relevant physiology and then expanding through the cultural and social determinants of behaviour. Professor Murphy does not hesitate to use frequent quotations from writers and poets to illustrate his exposition, and in spite of a few equivocal instances, the net result seems to enhance the general presentation. The 99 diagrams, photographs, and colour reproductions are of good quality, matching the paper, type, and binding. There is a 19-page combined glossary and index, and the suggested readings at the end of each chapter are additional to footnote references to relevant books and journal articles.

This is a textbook for the interested and not-too-sophisticated reader who hopes to become more interested without becoming at the same time more sophisticated. It cannot convince those who have already made up their minds that psychology is a l[illegible] hullabaloo about matters of common sense, but it m[illegible]y

do something to reassure those who had been led to believe that "it's all a matter of sex", or for that matter, of conditioned reflexes. This reviewer has only one major regret—he knows of no comparable introductory text written by a British psychologist.

ALASTAIR HERON

Social Psychology. By W. J. H. Sprott. (Pp. xiv + 208. 20*s*. net.) London: Methuen & Co. 1952.

In the past textbooks on social psychology, nearly all American, were content to serve up a large but often rather disconnected body of empirical findings. During the last few years social psychologists, becoming dissatisfied with such patchwork efforts, have attempted to produce something more systematic. The result was rather curious: a number of different treatises have appeared, mostly claiming to provide "an integrated body of theory and data". Unfortunately such theories are often organized around different sets of principles, selected according to the authors' personal preferences. For instance, whilst one recent book treats man entirely as a "language-manipulating animal", another barely mentions language. The example is, of course, extreme, yet symptomatic of the deficiency of present-day social psychology. There are too many "facts" and not enough unifying concepts for relating the facts within an agreed theoretical framework.

Professor Sprott therefore had a difficult task, which he accomplished most successfully. Without attempting premature system-building, which can only be achieved by leaving out inconvenient material, he presents the most important theories and empirical findings in a highly condensed but coherent manner, doing justice to a variety of complementary modes of approach.

The book is divided into three parts. The first, dealing with "the study of groups", begins with a useful discussion of notions like "culture", "personality", or "social class". Such words are an endless source of confusion when taken as referring to concrete entities. Students tend to cling to a rigid definition of a term like "social class", and are apt to despair if they happen to come across another inconsistent one. Sprott's philosophical background enables him to give a lucid account of the logical status of such terms. He explains that they are models constructed for definite but limited purposes out of the raw material of specific social situations. Such models approximate social reality in varying degrees, without ever completely encompassing it.

This is followed by two chapters on different types of groups, from such general concepts as "mass society" down to experimental studies of face-to-face groups, on which much research is going on just now. Lewin's field theory and Moreno's sociometry are outlined.

The chapter on leadership is somewhat disappointing, being too much concerned with the classification of different kinds of leaders and leaving out such important work as that of Helen Jennings and Ferenc Merei. The remainder of Part I summarizes studies of public opinion and attitude research.

Part II sets out with a description of social determinants in fields that were once thought to be the exclusive prerogative of general psychology. (Incidentally, it is demonstrated here how tenuous the distinction between "general" and "social" has become.) Among these is the concept of the self, where use is made of the theories of G. H. Mead, widely known in the United States but not easily accessible in this country; perception and memory are dealt with, and one of the best sections covers the controversial issue of intelligence. On this "battlefield" Sprott does not take sides, except for recommending that environmental factors should be explored before we fall back upon "innate endowment".

When it comes to the analysis of personality development in culture and the concept of national character, the author's skill in expounding complex theories becomes most apparent. Social psychologists and cultural anthropologists are today agreed that the first few years of life lay the foundation of the adult personality. There is, however, a certain amount of disagreement as to the exact way in which this happens. Sprott accepts the general approach of Linton and Kardiner, showing how it fits in with economic determinism in the wider sense. He has the courage to acknowledge our debt to Marx.

In view of the immature state of social psychology it was perhaps inevitable that the last part of the book, concerned with applications, should be the weakest. In the section on industry the Hawthorne studies, uncritically reported, loom largest. On the other hand there is some reference to the work of Georges Friedmann, whose work in industrial psychology deserves to be more widely known in this country.

The section on psychological medicine contains an interesting discussion of the vexed question of the criteria of "normality". There is also a brief description of various forms of group therapy.

With a book covering such a vast field one can hardly complain if some of one's own favourites have been left out. Perhaps the only serious omission is that of language and other forms of symbolic behaviour, which are increasingly coming into prominence.

Altogether Professor Sprott has produced an admirably clear survey of the fundamental tenets and data of social psychology. Although each page is closely packed, the style is easy, with a minimum of jargon; and all this has been accomplished in about half the usual length of books on social psychology. With these virtues it would be surprising if it did not become the standard introductory text in this country.

G. JAHODA

Psychology for Nurses. By Bess V. Cunningham. Second edition. (Pp. xi + 382. 26*s*.) New York: Appleton-Century-Crofts, Inc. 1951.

Unlike many second editions, this is an improvement on the first, published in 1946. Dr. Cunningham, who is Professor of Education in the University of Toledo, Ohio, has apparently not been content to provide the conventional "few lectures in psychology for the

student nurses", and has studied their needs with increasing sympathy and discernment. In the preface to the first edition she explained that for several years she had taught psychology to "student nurses from five local hospitals, who have come to the university for the first sixteen weeks of their academic training". One cannot help reflecting on the good fortune of these young ladies of Toledo: first in being saved from the fate of learning about psychology from the average sister-tutor, herself inadequately trained; secondly in receiving their first impressions of the subject *before* the rest of the nursing curriculum has made its mark; and thirdly in meeting with a university teacher who makes a sincere attempt to use the findings of education and psychology in her own methods.

To British readers the book may at times seem American in style and content, but it is much less so than might have been expected, and the clarity of exposition generally compensates for this. Though her references are mainly to books and papers published in the U.S.A., her choice is eclectic and up-to-date. Every chapter is provided with a summary, suggested activities likely to provide applications of the theoretical content, and a short bibliography for suggested reading, briefly annotated. She also provides a formal list of "References Cited in this Chapter", which might well have been dispensed with or brought together at the end of the book, as the practice leads to frequent multiple entries of the same books and papers. The book is provided with a useful, non-technical glossary of unfamiliar terms, and adequate author and subject indexes. Although the orientation throughout is toward the nurse, this text can be recommended to medical students and to more senior people such as industrial medical officers, requiring a simple but scientifically-based primer in modern psychology. ALASTAIR HERON

Eyes in Industry. By D. A. Campbell, W. J. B. Riddell, and A. S. McNalty. (Pp. 227 plus index; illustrated. 30*s*. net.) London: Longmans, Green. 1951.

Dorothy Campbell and her associates have written a most useful and basic treatise on the anatomy and physiology of the eye and its relationship to industry. It will be useful to personnel directors, safety engineers and management, and ophthalmologists not familiar with industrial problems. With that special genius that the British have, the authors have made the text lucid and complete, binding the complicated angles into a single integrated whole.

It is strange that she in Britain and I in the United States should produce books, the titles of which are so alike. There, however, the similarity ends. Dorothy Campbell has done everything that I wanted to do but could not, namely, make the basic functioning of the eye understandable to anyone reading it. Her diagrams, photographs, and her definitions of even the most technical material, are well done. There are things she stresses that we in this country should stress and have not, for instance, the care of adolescent eyesight and its relationship to industry.

Professor W. J. Riddell has written the second part of the book on visual hazards, injuries in industry, and their prevention. The important chemical research of the British war period is reflected. Miner's nystagmus, non-existent in the United States, has been the subject of British research for a long time. Most of the evidence seems to suggest that the absence of this disease in the U.S.A. is due to better lighting and a greater feeling for safety, and that miner's nystagmus is basically a psychomatic problem, but the final word has not yet been said. Britain, fortunately, was not a victim of epidemic kerato-conjunctivitis as we were and are still in some areas of this country, so they have nothing to contribute to this ever threatening problem. Sir Arthur McNalty has written part three on special aspects, injuries, and psychology of sight.

Research in visual testing in industry, the perfection of the equipment for this and the study and interpretation of the results, has hardly even started in Britain while it has made tremendous advances in the States. Elaborate eye protective equipment such as has been promoted in this country is not available in England.

That the authors do not discuss the relationship of the professional eye man with plant management and personnel reflects the loose character of their arrangements. Also, no mention is made of what we consider to be vitally important, namely the need for carefully outlined first aid technique and standing orders for nurses. While visiting British plants and talking to their industrial nurses I felt there had been too much leeway given to nurses. I am sure that their legal status does not include the practice of medicine, i.e. working and prescribing other than directly under defined doctor's orders. In the States we are very strict and our nurses are glad of the protection.

To anyone who wants to and needs to understand physiological and psychological visual functions and their relationship to industry this excellent book will be valuable. HEDWIG S. KUHN

BOOKS RECEIVED

(Review in a later issue is no. precluded by notice here of books recently received.)

A Catalogue of Film Strips for Health Education. Edited by John Burton. (Pp. 54. 2*s*. 6*d*.) London: The Central Council for Health Education. 1952.

Human Nature. Its Development, Variations and Assessment. By J. C. Raven. (Pp. 226. 12*s*. 6*d*.) London: H. K. Lewis. 1952.

Air Pollution. Proceedings of the United States Conference on Air-Pollution. By Louis C. McCabe, Chairman. (Pp. 847; illustrated. £5 6*s*. 6*d*.) London: McGraw-Hill Book Company Ltd. 1952.

Report on the Health of the Army, 1946–1948. (Pp. 116; 32 tables. No price.) London: The War Office. 1952.

The Scientific Study of Personality. By H. J. Eysenck. (Pp. 320 including index; illustrated. 30*s*.) London: Routledge & Kegan Paul. 1952.

The Young Delinquent in His Social Setting. By Thomas Ferguson. (Pp. 152 plus index; 21 figures, 79 tables. 10*s*. 6*d*.) Oxford University Press (London: Geoffrey Cumberlege). 1952.

The Nuffield Foundation. Seventh Report. (Pp. 161. No price.) Oxford University Press. 1952.

Occupational Health Prize

The Council of the British Medical Association is prepared to consider the award of an occupational health prize in the year 1953. The prize consists of a certificate and a money award to the value of £50.

The prize is established by the Council of the Association to encourage interest and research in the field of occupational health.

The prize will be awarded biennially.

Any member of the Association who is engaged in the practice of occupational health, either whole-time or part-time, is eligible to compete for the prize. Candidates may select their own subject.

The essay submitted must include personal observations and experiences collected by the candidate in the course of his work. If no essay entered is of sufficient merit no award will be made. Candidates in their entries should confine their attention to their own observations rather than to comments on previously published work on the subject, though reference to current literature should not be omitted when it bears directly on their results, their interpretations, and their conclusions.

Essays, or whatever form the candidate desires his work to take, must be sent to the Secretary, British Medical Association, B.M.A. House, Tavistock Square, London, W.C.1, not later than December 31, 1952.

No study or essay that has been published in the medical press or elsewhere will be considered eligible for the prize, and a contribution offered in one year cannot be accepted in any subsequent year unless it includes evidence of further work. A prizewinner in any year is not eligible for a second award of the prize.

If any question arises in reference to the eligibility of the candidate or the admissibility of his or her essay, the decision of the Council on any such point shall be final.

Preliminary notice of entry for this competition is required on a form of application to be obtained from the Secretary.

Each essay must be typewritten or printed on one side of the paper only, must be distinguished by a motto, and must be accompanied by a sealed envelope marked with the same motto and enclosing the candidate's name and address.

Inquiries should be addressed to the Secretary.

Scholarships in Aid of Scientific Research

The Council of the British Medical Association is prepared to receive applications for research scholarships, as follows:

An Ernest Hart Memorial Scholarship of the value of £250

A Walter Dixon Scholarship of the value of £250

One or More Research Scholarships each of the value of £200

These scholarships are given to candidates whom the Science Committee of the Association recommends as qualified to undertake research in any subject (including State medicine) relating to the causation, prevention, or treatment of disease.

Each scholarship is tenable for one year, beginning on October 1, 1953. A scholar may be re-appointed for not more than two additional terms. A scholar is not necessarily required to devote the whole of his or her time to the work of research, but may be a member of H.M. Forces or may hold a junior appointment at a university, medical school or hospital, provided the duties of such appointment will not, in the opinion of the Science Committee, interfere with his or her work as a scholar.

Applications for scholarships must be made not later than March 31, 1953, on the prescribed form, a copy of which will be supplied by the Secretary on application.

Applicants are required to furnish the names of three referees who are competent to speak as to their capacity for the research contemplated.

ABSTRACTS

(This section of the JOURNAL *is published in collaboration with the abstracting journal,* Abstracts of World Medicine, *published by the British Medical Association. The abstracts are divided into the following sections: toxicology; industrial physiology; industrial lung disease; industrial skin diseases; accidents and orthopaedic surgery; industrial ophthalmology; environment; general. Not all sections will necessarily be represented in any one issue)*

INDUSTRIAL TOXICOLOGY

Current Concepts of Beryllium Poisoning. ORDSTRAND, H. S. VAN (1951). *Ann. intern. Med.*, **35**, 1203.

In this review of the subject the clinical aspects of beryllium poisoning are discussed and are illustrated by reference to the author's series of 450 cases. A brief description of the properties and uses of beryllium and its compounds is given.

Beryllium causes two types of skin lesion. The commonest manifestation is a contact dermatitis, which is eczematous in type and has been shown to be due to hypersensitivity. The skin of affected individuals gives a positive reaction to patch tests with high dilutions of beryllium compounds. The second type of skin lesion is a chronic granuloma, which appears to be a local reaction following accidental implantation of beryllium compounds at the site of an abrasion. The granuloma is cured by excision.

The respiratory tract may exhibit an acute reaction to beryllium and its compounds, which may consist in minor upper respiratory symptoms or in a pneumonitis which has been called "acute berylliosis". The latter condition begins with cough and dyspnoea, and after 2 or 3 weeks radiological examination shows symmetrical infiltration of both lung fields. The author had 98 cases of this type, 12 of which ended fatally. Death occurs about 30 days after the onset of the illness, and is due to asphyxia with or without acute cor pulmonale. In the non-fatal cases complete recovery occurs within 4 months, and over a 10-year period there has been no evidence that the chronic type of pulmonary disease supervenes in the recovered cases. Hyperglobulinaemia and diminished liver function have been demonstrated during the acute illness, and pathological studies show an intra-alveolar odematous exudate in the lungs, with centrilobular necrosis in the liver.

The chronic form of respiratory involvement appears some time after exposure, even up to 10 years later. Radiological changes, consisting of disseminated fine nodular infiltration, precede the development of symptoms. The latter are similar to those of acute berylliosis with the addition of anorexia and weight loss, and intermittent fever occurs in a few cases. Though the chronic disease does not progress so rapidly as the acute form, the outlook is sombre. One-third of the author's patients died and another third showed steady deterioration. The pathological changes affect the interstitial tissues of the lungs, and consist of a chronic, sarcoid-like granulomatous infiltration. Beryllium has been found in renal calculi, lymph nodes, and the liver. Death is due to chronic cor pulmonale.

The conclusion reached by both Machle and Hardy that beryllium poisoning is a systemic disease is accepted, and some discussion follows on the possible physiopathological mechanisms involved. Although toxic effects in the lung, liver, and kidneys of animals have been produced, nothing resembling chronic berylliosis in man has been seen. Gardner and others have produced osteogenic sarcomata in rabbits by injecting beryllium compounds, but such lesions have not been found in man. The radioactive isotope ^{7}Be has been shown to enter the bones of small animals, and this fact has suggested the hypothesis that chronic berylliosis may occur as a result of the release of beryllium from the bones following various stresses occurring at a later date. The observation by Curtis that the acute eczematous contact dermatitis is due to hypersensitivity has led to the suggestion that acute berylliosis may have a similar aetiology. Other experimental observations have shown that beryllium replaces magnesium in various enzyme systems which subsequently become inhibited, and this has been adduced as evidence that the manifestations of chronic berylliosis may be due to widespread cellular changes.

Although ACTH and cortisone have been shown to be of great benefit in treatment of chronic berylliosis, emphasis is laid on the importance of engineering methods of control of beryllium exposure in industry.

W. K. S. Moore.

Treatment of Cyanide Poisoning in Industry. WOLFSIE, J. H. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 417.

This is an important report of 12 cases of cyanide poisoning, chosen as typical of the severe intoxication met with from time to time in a chemical plant where large volumes of hydrogen cyanide and cyanide compounds and derivatives are manufactured, and where there has been no death from cyanide poisoning during the last 30 years. The men all recovered completely. One had lost consciousness, but had not stopped breathing; 10 were apnoeic, in coma; and 1 was convulsed.

The most effective treatment is the intravenous administration of sodium nitrite and sodium thiosulphate; this, however, was not applied immediately. After rescue and removal to fresh air, immediate essential treatment is: (1) artificial respiration, continued without interruption; (2) inhalations of amyl nitrite, repeated

The Scientific Study of Personality. By H. J. Eysenck. (Pp. 320 including index; illustrated. 30s.) London: Routledge & Kegan Paul. 1952.

The Young Delinquent in His Social Setting. By Thomas Ferguson. (Pp. 152 plus index; 21 figures, 79 tables. 10s. 6d.) Oxford University Press (London: Geoffrey Cumberlege). 1952.

The Nuffield Foundation. Seventh Report. (Pp. 161. No price.) Oxford University Press. 1952.

Occupational Health Prize

The Council of the British Medical Association is prepared to consider the award of an occupational health prize in the year 1953. The prize consists of a certificate and a money award to the value of £50.

The prize is established by the Council of the Association to encourage interest and research in the field of occupational health.

The prize will be awarded biennially.

Any member of the Association who is engaged in the practice of occupational health, either whole-time or part-time, is eligible to compete for the prize. Candidates may select their own subject.

The essay submitted must include personal observations and experiences collected by the candidate in the course of his work. If no essay entered is of sufficient merit no award will be made. Candidates in their entries should confine their attention to their own observations rather than to comments on previously published work on the subject, though reference to current literature should not be omitted when it bears directly on their results, their interpretations, and their conclusions.

Essays, or whatever form the candidate desires his work to take, must be sent to the Secretary, British Medical Association, B.M.A. House, Tavistock Square, London, W.C.1, not later than December 31, 1952.

No study or essay that has been published in the medical press or elsewhere will be considered eligible for the prize, and a contribution offered in one year cannot be accepted in any subsequent year unless it includes evidence of further work. A prizewinner in any year is not eligible for a second award of the prize.

If any question arises in reference to the eligibility of the candidate or the admissibility of his or her essay, the decision of the Council on any such point shall be final.

Preliminary notice of entry for this competition is required on a form of application to be obtained from the Secretary.

Each essay must be typewritten or printed on one side of the paper only, must be distinguished by a motto, and must be accompanied by a sealed envelope marked with the same motto and enclosing the candidate's name and address.

Inquiries should be addressed to the Secretary.

Scholarships in Aid of Scientific Research

The Council of the British Medical Association is prepared to receive applications for research scholarships, as follows:

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A Walter Dixon Scholarship of the value of £250

One or More Research Scholarships each of the value of £200

These scholarships are given to candidates whom the Science Committee of the Association recommends as qualified to undertake research in any subject (including State medicine) relating to the causation, prevention, or treatment of disease.

Each scholarship is tenable for one year, beginning on October 1, 1953. A scholar may be re-appointed for not more than two additional terms. A scholar is not necessarily required to devote the whole of his or her time to the work of research, but may be a member of H.M. Forces or may hold a junior appointment at a university, medical school or hospital, provided the duties of such appointment will not, in the opinion of the Science Committee, interfere with his or her work as a scholar.

Applications for scholarships must be made not later than March 31, 1953, on the prescribed form, a copy of which will be supplied by the Secretary on application.

Applicants are required to furnish the names of three referees who are competent to speak as to their capacity for the research contemplated.

ABSTRACTS

(This section of the JOURNAL *is published in collaboration with the abstracting journal,* Abstracts of World Medicine, *published by the British Medical Association. The abstracts are divided into the following sections: toxicology; industrial physiology; industrial lung disease; industrial skin diseases; accidents and orthopaedic surgery; industrial ophthalmology; environment; general. Not all sections will necessarily be represented in any one issue)*

INDUSTRIAL TOXICOLOGY

Current Concepts of Beryllium Poisoning. ORDSTRAND, H. S. VAN (1951). *Ann. intern. Med.*, **35**, 1203.

In this review of the subject the clinical aspects of beryllium poisoning are discussed and are illustrated by reference to the author's series of 450 cases. A brief description of the properties and uses of beryllium and its compounds is given.

Beryllium causes two types of skin lesion. The commonest manifestation is a contact dermatitis, which is eczematous in type and has been shown to be due to hypersensitivity. The skin of affected individuals gives a positive reaction to patch tests with high dilutions of beryllium compounds. The second type of skin lesion is a chronic granuloma, which appears to be a local reaction following accidental implantation of beryllium compounds at the site of an abrasion. The granuloma is cured by excision.

The respiratory tract may exhibit an acute reaction to beryllium and its compounds, which may consist in minor upper respiratory symptoms or in a pneumonitis which has been called "acute berylliosis". The latter condition begins with cough and dyspnoea, and after 2 or 3 weeks radiological examination shows symmetrical infiltration of both lung fields. The author had 98 cases of this type, 12 of which ended fatally. Death occurs about 30 days after the onset of the illness, and is due to asphyxia with or without acute cor pulmonale. In the non-fatal cases complete recovery occurs within 4 months, and over a 10-year period there has been no evidence that the chronic type of pulmonary disease supervenes in the recovered cases. Hyperglobulinaemia and diminished liver function have been demonstrated during the acute illness, and pathological studies show an intra-alveolar odematous exudate in the lungs, with centrilobular necrosis in the liver.

The chronic form of respiratory involvement appears some time after exposure, even up to 10 years later. Radiological changes, consisting of disseminated fine nodular infiltration, precede the development of symptoms. The latter are similar to those of acute berylliosis with the addition of anorexia and weight loss, and intermittent fever occurs in a few cases. Though the chronic disease does not progress so rapidly as the acute form, the outlook is sombre. One-third of the author's patients died and another third showed steady deterioration. The pathological changes affect the interstitial tissues of the lungs, and consist of a chronic, sarcoid-like granulomatous infiltration. Beryllium has been found in renal calculi, lymph nodes, and the liver. Death is due to chronic cor pulmonale.

The conclusion reached by both Machle and Hardy that beryllium poisoning is a systemic disease is accepted, and some discussion follows on the possible physiopathological mechanisms involved. Although toxic effects in the lung, liver, and kidneys of animals have been produced, nothing resembling chronic berylliosis in man has been seen. Gardner and others have produced osteogenic sarcomata in rabbits by injecting beryllium compounds, but such lesions have not been found in man. The radioactive isotope ^{7}Be has been shown to enter the bones of small animals, and this fact has suggested the hypothesis that chronic berylliosis may occur as a result of the release of beryllium from the bones following various stresses occurring at a later date. The observation by Curtis that the acute eczematous contact dermatitis is due to hypersensitivity has led to the suggestion that acute berylliosis may have a similar aetiology. Other experimental observations have shown that beryllium replaces magnesium in various enzyme systems which subsequently become inhibited, and this has been adduced as evidence that the manifestations of chronic berylliosis may be due to widespread cellular changes.

Although ACTH and cortisone have been shown to be of great benefit in treatment of chronic berylliosis, emphasis is laid on the importance of engineering methods of control of beryllium exposure in industry.

W. K. S. Moore.

Treatment of Cyanide Poisoning in Industry. WOLFSIE, J. H. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 417.

This is an important report of 12 cases of cyanide poisoning, chosen as typical of the severe intoxication met with from time to time in a chemical plant where large volumes of hydrogen cyanide and cyanide compounds and derivatives are manufactured, and where there has been no death from cyanide poisoning during the last 30 years. The men all recovered completely. One had lost consciousness, but had not stopped breathing; 10 were apnoeic, in coma; and 1 was convulsed.

The most effective treatment is the intravenous administration of sodium nitrite and sodium thiosulphate; this, however, was not applied immediately. After rescue and removal to fresh air, immediate essential treatment is: (1) artificial respiration, continued without interruption; (2) inhalations of amyl nitrite, repeated

every 5 minutes for 25 minutes; (3) removal of contaminated clothing and cleansing of any contaminated skin with soap and water; and (4) recumbency and warmth. Four men recovered with this treatment alone; 2 received in addition intravenous sodium nitrite, 15 minutes and 45 minutes respectively after their collapse; 1 received in addition intravenous sodium thiosulphate, 15 minutes after his collapse; 5 were given the standard intravenous sodium nitrite–thiosulphate therapy at intervals from the time of collapse varying from 40 to 80 minutes. (Heartening news to those who have been advised: "You have only about 2 minutes to do anything".)

If the poison has been ingested and the patient is conscious, vomiting should be induced or the stomach washed out by the physician, and this should be followed by the oral administration of 1 pint (568 ml.) of 1% solution of sodium thiosulphate, which should be repeated in 15 minutes.

It is insisted that where cyanides are handled all personnel must be well trained in the administration of artificial respiration and the first-aid measures required, and that a cyanide first-aid outfit must be kept ready and at hand. *M. A. Dobbin Crawford.*

Aldrin Poisoning in Man. Report of a Case. SPIOTTA, E. J. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 560.

This is a detailed report of the first case on record of poisoning by ingestion of "aldrin" (hexachlorohexahydrodimethanolnaphthalene; $C_{12}H_8Cl_6$). ("Chlordane" and "dieldrin" are related compounds.) The patient, a man of 23, who had taken 6 oz. (170 ml.) of a diluted mixture of aldrin with suicidal intent, completely recovered.

The quantity of the insecticide ingested was estimated as 1·8 g., or 25·6 mg. per kg. of the patient's weight. Absorption was extremely rapid. Gastric lavage with magnesium sulphate solution within 20 minutes produced no evidence of aldrin content. The effects of intoxication were a swift irritation of the cerebral cortex and acute renal injury. General convulsions began while the stomach tube was being passed, and were controlled only by heavy sedation. Before the patient was admitted to a local hospital, barbiturates, morphine sulphate, and atropine sulphate were given intramuscularly in rather large doses (quantities not stated). He was then transferred to the Kennedy Hospital, Tennessee. During the first 24 hours after his admission he received 0·4 g. (6 gr.) of phenobarbitone sodium intramuscularly and 1·5 g. (22½ gr.) of amylobarbitone sodium intravenously. Fairly heavy sedation with the barbiturates was continued for a week, and then these drugs were replaced by paraldehyde, given (route not mentioned) in 10-ml. doses every 2 to 4 hours. Paraldehyde was continued until the 12th day of the illness and then stopped, the patient having become lucid, oriented, and quieter.

At the time of the patient's admission to the Kennedy Hospital he had gross haematuria and marked albuminuria. Fluids were restricted for 24 hours and then given freely. On the 18th day after he had taken the poison his urine became normal except for a low specific gravity. Liver function tests gave normal results, with the exception on one occasion of the icterus index, which on the 4th day was found to be 15 units. At this time the non-protein nitrogen content of the blood was estimated as 81 mg. per 100 ml. There was no sign of the liver necrosis which has been found in animals poisoned with aldrin (and which is so grave a feature in poisoning with other chloronaphthalene compounds, such as "seekay" wax in human beings). Neither in this patient nor in experimental animals was there any significant alteration in the blood count or in the bone-marrow picture.

Encephalographic tracings are reproduced and biochemical findings given in detail.

M. A. Dobbin Crawford.

Toxicity of an Imidazoline (or Glyoxalidine) Fungicide. CARPENTER, C. P., WEIL, C. S., and SMYTH, H. F. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 494.

Benzidine (4 : 4′-Diaminobiphenyl) and Substituted Benzidines. A Microchemical Screening Technic for Estimating Levels of Industrial Exposure from Urine and Air Samples. GLASSMAN, J. M., and MEIGS, J. W. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 519.

INDUSTRIAL LUNG DISEASES

A Study of Pneumoconiosis in Fluorspar Workings. LUTON, P., and CHAMPEIX, J. (1951). *Arch. Mal. prof.*, **12**, 505.

The main purpose of this paper is to draw attention to a serious risk of pneumoconiosis among workers in fluorspar mines in France, 2 of which were studied by the authors in detail. At the first there were 34 underground workers who had never been exposed to any other form of dust and were all concerned with mineral extraction and rock drilling, 19 of whom showed radiological evidence of pneumoconiosis. Details are given of 10 of these cases, selected because of the severity of the pneumoconiosis or the rapidity with which it developed.

There were 7 cases of severe pneumoconiosis which had developed after less than 2 years' exposure. One of these patients developed massive radiographic opacities with a negative sputum and died at the age of 29, 8 years after leaving dust exposure. Another patient with a negative sputum was treated with streptomycin and PAS without any effect. Another had a positive sputum and was treated by pneumothorax and with streptomycin and PAS, and despite this the lung radiograph showed some increase in nodulation. Three more fatal cases with severe pneumoconiosis (and without bacteriological evidence of tuberculosis) after more than 2 years' exposure are described, in 1 of which the patient died at the age of 23 having had only 2½ years' exposure. At the second mine studied, out of 31 underground workers only 11 had abnormal radiographic appearances and none of the films showed typical pneumoconiosis [although they are described as showing micronodulation]. Incomplete radiological surveys were carried out

at 2 other mines, at 1 of which were found some marked cases of pneumoconiosis similar to those at the first undertaking.

The methods used in all the mines studied were essentially similar, drilling and shot firing being carried out both in the seam and also in the surrounding rock, which contained 60% to 96% free silica. Dust samples were taken by aspirating through a tetrachloronaphthalene filter, dissolving the filter, and counting the particles. The results of dust counts from 4 different workings are given, and show concentrations varying from 1,800 to 500,000 particles less than 10μ in diameter per ml., with a free silica content of 6% to 58%. In view of the very different incidence of severe pneumoconiosis noted among workers at the first 2 mines, these undertakings were studied more closely. It was found that at the first the seam of fluorspar was thinner, with a silica content of 60%, whereas at the second there was a wider seam with a silica content of 0·8 to 5%. There was more rock work in the first mine, and the workers worked more often with dry drills.

Finally, the authors speculate on the possible causes of the rapid development of pneumoconiosis in the first group of workers studied. They suggest that the atypical radiological appearances in workers in the second mine may have been due to the high content of fluorspar dust causing inflammation in the lungs, and that in the first the effect of the high silica content may have been accelerated by this supposed inflammatory action of fluorspar dust, resulting in the rapid development of the disease. [It is not clear why the authors are dissatisfied with the obvious explanation that there were higher dust concentrations with a higher silica content in the first mine.] *C. M. Fletcher.*

Silicosis in the Ball-clay and China-clay Industries. THOMAS, R. W. (1952). *Lancet*, **1**, 133.

The production of ball clay and china clay in mines and quarries is not a dusty occupation, and there is no evidence that workers engaged in these mines and quarries have ever suffered from pneumoconiosis. On the other hand, the preparation of these materials for use in industry by grinding and bagging for transport may expose workers to high concentrations of dust; but hitherto no reports of pneumoconiosis from these causes have been reported. Two such cases are now presented.

The first man died at the age of 64. He had spent nearly all his working life in the ball-clay industry, much of it in drying-kilns and grinding-mills. He developed shortness of breath, and on radiological examination a picture suggesting nodular silicosis was seen. Necropsy revealed classical silicotic nodulation, mostly in the upper parts of the lungs, without tuberculosis. The second case was in a china-clay worker. His industrial history was not known precisely, but for 5 years before death he had been employed in trucking dry china clay from the drying-kilns to the mill-house. He had also been concerned with the grinding of china stone and felspar many years previously. He died suddenly. At necropsy tuberculo-silicotic masses with moderate numbers of small classical silicotic nodules were found.

It is clear that the silicosis in the first case was attributable to ball clay, which may contain up to 29% free silica. The silico-tuberculosis of the second case cannot be definitely attributed to china-clay dust, for the man had had a mixed industrial history.

The author suggests that further investigation is required of men exposed to china-clay and ball-clay dusts. *C. M. Fletcher.*

Report on a Country-wide Study of Silicosis in the Metal Mines of Japan. YAMAMOTO, M. (1951). *Arch. Industr. Hyg. occup. Med.*, **3**, 339.

Etiologic Factors in Bronchiogenic Carcinoma with Special Reference to Industrial Exposures. Report of Eight Hundred and Fifty-seven Proved Cases. WYNDER, E. L., and GRAHAM, E. A. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 221.

The patient's occupational history was obtained in 857 proved cases of bronchogenic carcinoma by personal interview. The patients included 803 males, of whom 43 had adenocarcinoma and the rest epidermoid, undifferentiated, or unclassified carcinoma. The occupational distribution of the cases was determined, patients who had had several occupations being allocated to that one in which there was the greatest apparent exposure to irritative dusts or fumes, and in which they had spent at least 5 years during the last 40. For 200 of the patients, all from Barnes Hospital Chest Service, St. Louis, a control group of 200 patients of similar age and economic status suffering from other diseases of the chest was studied. Comparison of these 2 groups showed several suggestive (though not decisive) differences in occupational distribution, of which the higher proportion of metal workers and painters observed among the patients with carcinoma accords best with the findings of other workers. No systematic difference was observed in the proportion of cases from urban and rural communities. The authors discuss the difficulty of making a proper evaluation of occupational factors in the aetiology of lung cancer in view of the large number of controls required and the need to obtain a complete detailed occupational history.

The patients with bronchogenic carcinoma are also classified according to their smoking habits: 98·6% of the males and 56·2% of the females with epidermoid, undifferentiated or unclassified carcinoma were smokers. Among the cases of adenocarcinoma, 90·7% of the males and 9·1% of the females were smokers. The occupational distribution of the non-smokers and minimal smokers (less than 4 cigarettes a day, or the equivalent in tobacco, for 20 years) is given, with detailed occupational histories of the 11 male non-smokers. The authors suggest that a weakly carcinogenic factor associated with certain occupations may act synergistically with tobacco smoke to produce a greatly increased incidence of lung cancer. They suggest also that adenocarcinoma is more closely associated with exogenous irritation than they had previously concluded.

[The authors' summary contains a number of statements for which no evidence is presented in the text.]
P. D. Oldham.

INDUSTRIAL SKIN DISEASES

Clinical, Social, and Occupational Aspects of Industrial Dermatitis. HEWITT, M. (1951). *Lancet*, **2**, 1105.

Industrial dermatitis is one of the most serious occupational diseases and involves every branch of industry. Oil folliculitis, benign and malignant growths of the skin due to tar, chrome ulceration, and chloracne are easily recognized, but inflammation of the skin ascribed to dust, liquids, or vapour accounts for the majority of cases. The dermatitis appears on the exposed parts of the body and is aggravated by repeated exposure. It clears rapidly on removal of the irritant substance. There is hardly any substance that cannot produce sensitization. In the literature there is little attempt to distinguish between industrial dermatitis and constitutional eczema which happens to occur among workmen. Patients with industrial dermatitis are often found to have unsatisfactory work records and severe social and emotional difficulties.

In this paper is reported a study of 80 consecutive cases of industrial dermatitis of over 2 months' duration. There were 64 men and 16 women. The age incidence was the same as that for idiopathic eczema reported by MacCormac (*Brit. J. Derm. Syph.*, 1937, **49**, 490). Of the 80 patients 63 were married. Little information was obtained by attempts at classification on an occupational or dermatological basis, since associated social and emotional disturbance appears to play an integral part in the cause of the malady. There was no evidence that increased incidence could be attributed to new manufacturing processes. The rising incidence among miners, where the hazards have not materially altered, refutes this theory. The idea of the disease is often sown in the patient's mind by the appointed factory doctor's first opinion or by views expressed by industrial nurses, trade unions, or the man's workmates.

The study suggests that industrial irritants and sensitizers are aetiologically insignificant in comparison with the overriding social and occupational problems which these patients present. There appears to be a great gap between the statutory recognition of the disease and attempts to prevent it. Little benefit results from purely local treatment. The probable nature of his difficulties must be explained to the patient, who must be handled competently when his first fears become apparent. The increased incidence of the condition has been peculiarly associated with advances in social legislation designed for the public health. Many of the cases suggest that a lack of understanding of the problems of industrial dermatitis and incoordination between the medical and social services are encouraging ill-health which is not confined to the immediate sufferers. Compulsory notification of industrial dermatitis might be a preliminary helpful measure in forging a link between diagnostic and preventive industrial health services. A joint social, clinical, and occupational approach to the problem of industrial dermatitis appears to be the method most likely to reduce its incidence. *K. M. A. Perry.*

Occupational Dermatoses in Physicians. EPSTEIN, E. (1951). *J. Amer. med. Ass.*, **147**, 1751.

In view of the fact that physicians are exposed to many irritating chemicals and infections in their professional work, a questionnaire was sent to 75 dermatologists requesting information on the incidence of dermatoses in physicians under their care; 60 (80%) replied.

Contact dermatitis was the commonest condition reported. Mechanical trauma in scrubbing-up was the commonest cause of dermatitis of the hands, and although reduction of scrubbing time by the use of agents such as hexachlorophene prevented many cases of dermatitis, they were sometimes themselves responsible for sensitization. Mercurial antiseptics and formaldehyde (in pathologists) were frequent causes, as also were orris root used in certain glove powders and the rubber gloves themselves. Of local anaesthetics procaine was the commonest sensitizing agent, and of antibiotics penicillin was incriminated 5 times as often as streptomycin. However, self-medication was claimed to be a commoner cause of contact dermatitis in physicians than was occupational exposure.

Of infections, impetigo and furunculosis were the commonest, but dermatophytids were no commoner in physicians than in the general population. Parasitic infestations were uncommon, but 4 dermatologists had caught scabies from patients, and 8 thought they had been infected by patients with warts. Although, in the author's view, accidental syphilitic infection is rare, 16 contributors reported a total of 27 extragenital chancres in physicians. On the other hand, 22 contributors (40%) stated that they had never seen any kind of skin infection passed to a physician by a patient.

Radiodermatitis is the most serious skin disease found in physicians. Although efficient means of protection are available, too many cases still occur as the result of ignorance and carelessness. Injuries such as cuts at operations or burns from electrical appliances were uncommon. The author feels that more could be done by medical schools to teach students the importance of protecting themselves against occupational skin diseases.

Stephen G. Gang.

Occurrence of Contact Dermatitis from Oil Soluble Gasoline Dyes. LAMB, J. H., and LAIN, E. S. (1951). *J. invest. Derm.*, **17**, 141.

This is a report of 3 patients who suffered from contact dermatitis due to the azo-dyes used in colouring commercial petrol. The histories and patch-test investigations showed that the subjects were also sensitive to chemically related compounds. *John T. Ingram.*

Occupational Arsenical Dermatitis. A Study Among Employees at a Copper Ore Smelting Work Including Investigations of Skin Reactions to Contact with Arsenic Compounds. (In English.) HOLMQVIST, I. (1951). *Acta derm.-venereol., Stokh.*, **31**, Suppl. 26, 1. Bibliography.

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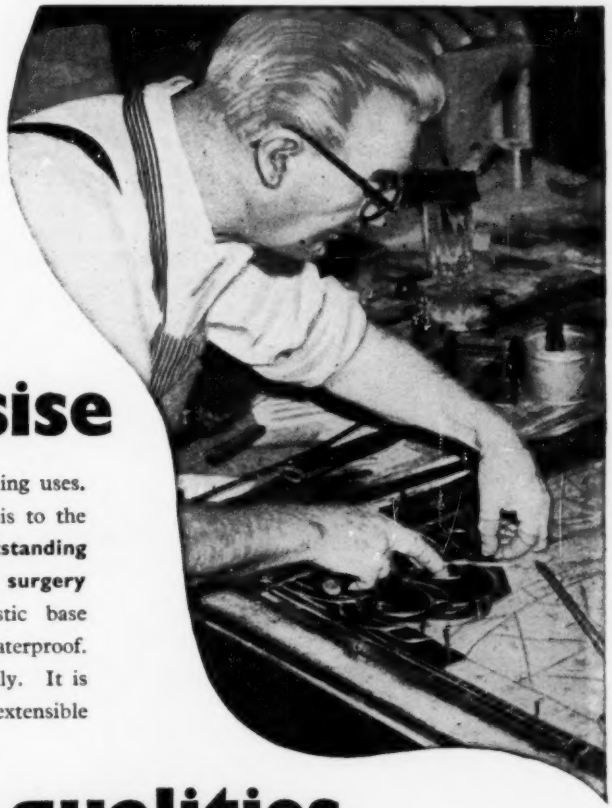

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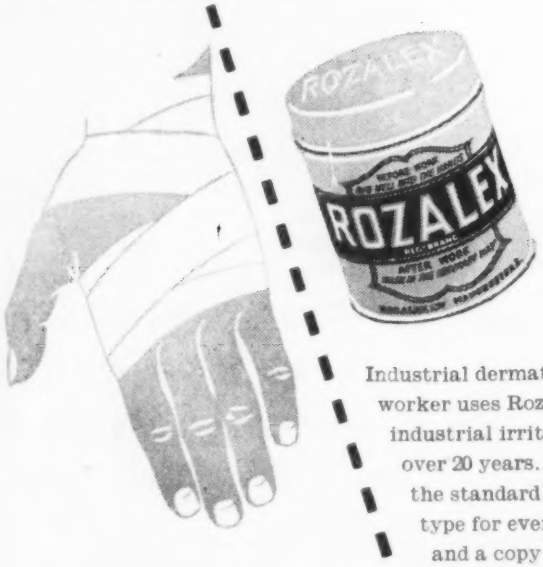

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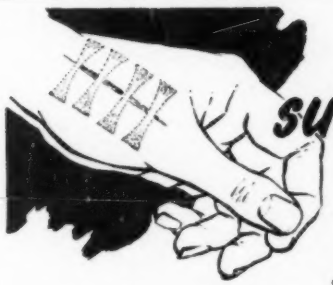

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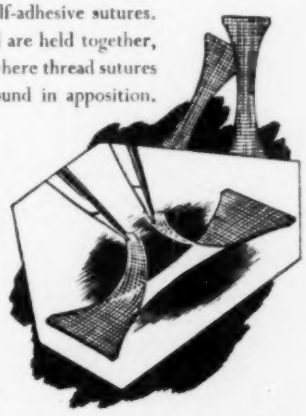

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